Eating behaviour and body weight in a sample of adult women : the role of stress and dietary restraint

Roberts, Clifford John

~~An investigation into the effects of stress on eating behaviour in healthy adult women~~

EATING BEHAVIOUR AND BODY WEIGHT IN A SAMPLE OF ADULT WOMEN: THE ROLE OF STRESS AND DIETARY RESTRAINT.

Cliff Roberts

Institute of Psychiatry
King's College London.

PhD Thesis

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ABSTRACT

This naturalistic longitudinal study examined the effects of stress on eating behaviour and changes in body weight. The study was conducted at a London University with 71 healthy women volunteers taking part. The aim of the study was to examine potential predictors of weight change initially by means of correlational analysis and t-tests and subsequently developing a model using regression analysis.

Predictors of weight change were hypothesised to be stress, dietary restraint, mental health, bingeing and changes in food choice. In response to an unseen written examination these potential predictors of weight change were assessed for mediator and moderator effects on weight change. Using regression analysis a model was developed demonstrating stress and dietary restraint as significant predictors of weight change.

A significant moderator of weight change was identified as the change in dietary restraint score during the stress period. Change in bingeing behaviour was seen as a significant mediator of the effects of restraint and change in cortisol secretion on weight change during the stress period. Change in food preferences towards a diet higher in saturated fat, carbohydrates and alcohol consumption mediated the effects of cortisol secretion during the stress period on weight change.

Other significantly correlated factors involved in weight change were identified as anxiety, depression, mastery and coping behaviour, with anxiety and depression reaching potentially pathological levels during the stress period.

Potential pathological symptoms of anxiety and depression were reported during the week of the examination, along with a reduction in mastery and coping behaviour. Findings from this study indicate that it would be of benefit to identify individuals susceptible to detrimental changes in mental health, mastery and coping skills during periods of increased stress, and offer them brief interventions aimed at maintaining mastery and coping skills. This may also reduce the tendency to switch to a diet high in saturated fats and refined carbohydrates, which is known to be involved with the development of peripheral vascular disease and type II diabetes.

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Chapter 1

1 Introduction: overview of the study:

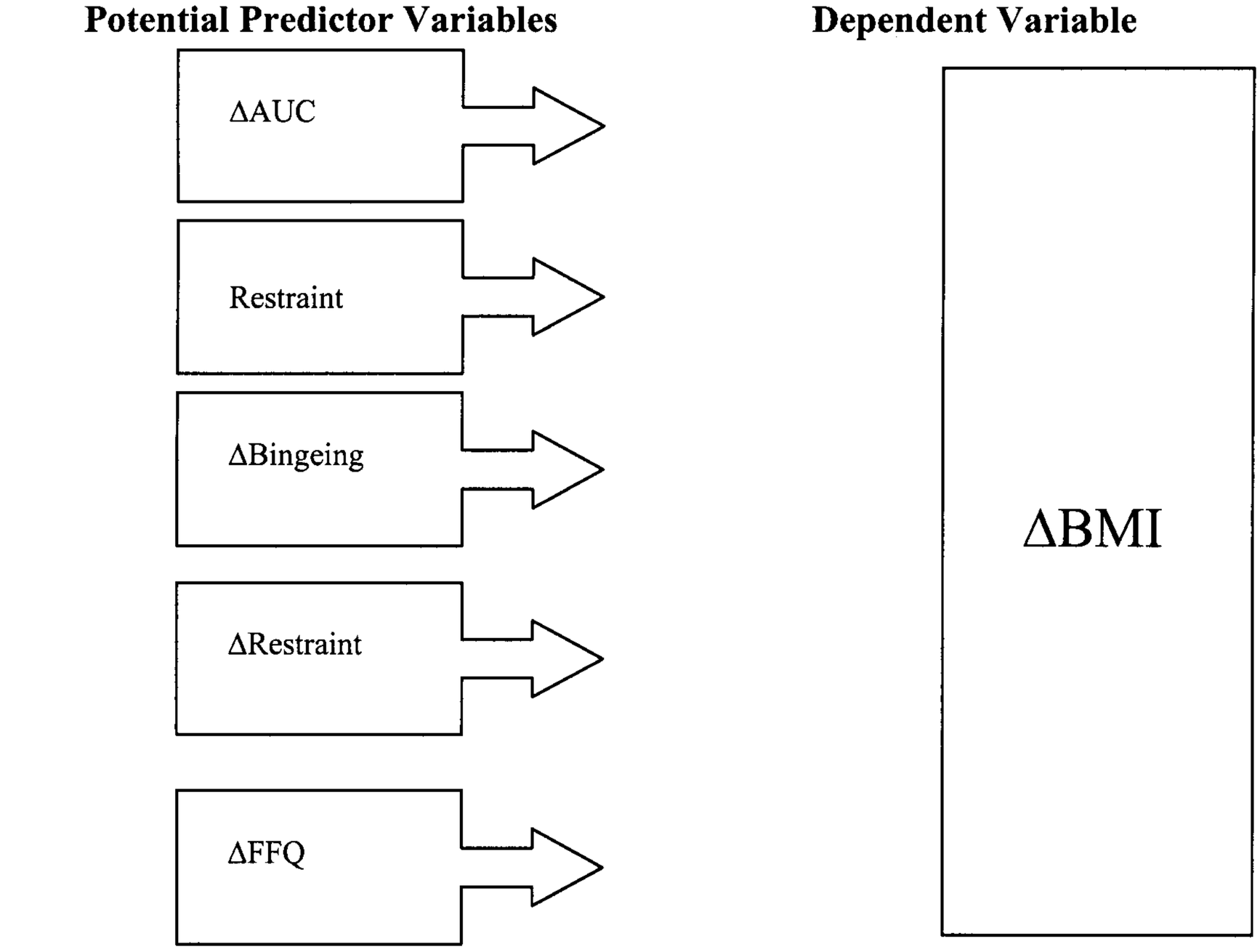
The relationship between stress, eating behaviour, and weight is complex and it is likely genetic, biological, psychological and sociocultural factors are involved. Research in this area has been conducted mostly within the laboratory environment, however, naturalistic studies are beginning to emerge. The laboratory environment lends itself to the testing of the General Effect Theory proposed by Greeno and Wing (1994), in that physiological variables such as cortisol or adrenaline secretion will generally alter in response to a stressor. This alteration of biological variables will result in a general response to stress. Due to the design of laboratory studies it is often difficult to relate findings to a naturalistic longitudinal environment, for example, the consumption of a few more/less grams of carbohydrate in response to a stressor does not provide evidence that body weight will change. Naturalistic longitudinal studies provide an opportunity to examine individual differences (Greeno and Wing 1994), in response to a stressor over time. Thus the effects of individual differences such as cortisol secretion, dietary restraint, bingeing behaviour, mood and food choice during times of stress can be examined in naturalistic studies. To date however the findings from such studies have not conclusively determined the effects of changes in these variables, on weight change during times of stress. The literature suggests that some individuals increase their caloric intake during times of stress and others reduce their intake (Epel et al 2001; Stone and Brownell 1994). The "comfort food" hypothesis proposes that a high carbohydrate and saturated fat diet is consumed during times of

stress to ameliorate the psychophysiological effects of stressors. This may be part of avoidance behaviour in order to reduce the impact of stress on the individual, i.e. "comfort food" may enhance mood, at least in the short term. Mastery and coping skills may also impact on cognitive decision making when choosing food type and the amount consumed. Individuals with low mastery and coping skills may be more likely to indulge in "comfort eating" in an attempt to reduce/ avoid the effects of stress. Subjects with a high level of dietary restraint during times of stress may become disinhibited, and as a result, an increase in bingeing behaviour will ensue. As a consequence of increased bingeing, daily carbohydrate and saturated fat consumption has been observed to rise in these individuals in response to a stressor but not in those low in dietary restraint. These changes in dietary intake particularly that of "comfort food" within the general population will have a significant impact on health. For example, a diet high in saturated fat is associated with peripheral vascular disease, and hypertension, whilst a diet high in simple carbohydrates is associated with Type II diabetes later in life (National Heart, Lung and Blood Institute, 1998). There may also be a subsequent increase in Body Mass Index (BMI) but it is not known if this is a transient or longer lasting effect. When bingeing is sustained over long periods it becomes a health risk leading to obesity (Prentice 2001), and the pathophysiology mentioned above. Obesity is a major problem in the western world and in the UK recent figures show that 23% of men and 25% of women were obese in 2002 (Rennie and Jebb 2005). The aim of this study was to examine individual differences, which may be predictors of change in Body Mass Index (ΔBMI), and develop a model (see page 20 & 21), which will identify, which predictors are mediators or moderators of ΔBMI. A greater understanding of individual differences associated with weight gain and the concurrent potential to develop obesity will provide information for policy

makers on the suitability of individualised interventions. For example, brief interventions aimed at enhancing mastery and coping skills coupled with dietary information will be more effective than general approaches such as pharmaco-therapeutics, slimming aids or the use of popular self selected diets, such as The Atkins Diet. Many popular diets, such as, The Atkins Diet, are unhealthy because they cut out food groups such as carbohydrates in preference for what are known to be unhealthy food groups such as saturated fat.

The diagram on the next page, shows potential predictor variables and the dependent variable, which are investigated in the study.

Fig:1.1 Potential predictor variables, that may have direct main effects on the dependent variable (ΔBMI).



Key

ΔAUC = Cortisol secretion

ΔBingeing = Change in bingeing behaviour

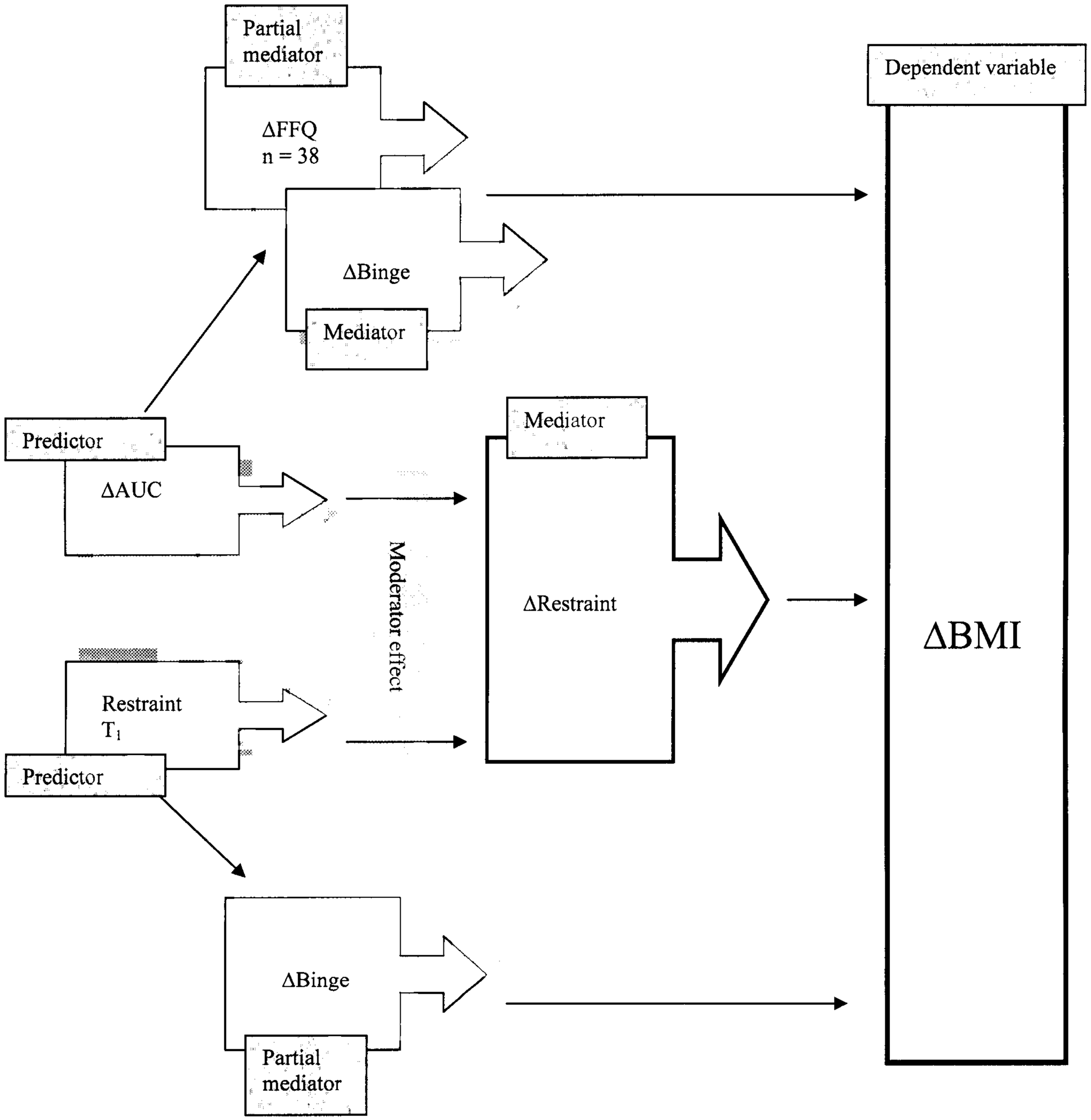
ΔRestraint = Change in dietary restraint

ΔFFQ = Change in food choice and consumption

ΔBMI = Change in BMI

The potential predictor variables were analysed using linear regression, identifying mediators and moderators of the effects of the predictors on change in BMI. See fig 1.2 below (page 21). This provides an overview of the model developed from the study.

Figure 1.2. Model supported by the findings of regression analysis to explain the relationship between predictors, mediators, partial mediators and the dependent variable ΔBMI. n = 71 unless stated.



Key.

Predictors ΔAUC, Restraint T1
Mediators/ partial mediators ΔFFQ, ΔBinge and ΔRestraint,
Dependent variable ΔBMI.

1.1) Hypotheses

At Base line:

1. Body Mass Index (BMI), will be correlated with dietary restraint and bingeing behaviour scores.

In response to the stressor:

2. Salivary cortisol excretion will be elevated and the increase will be associated with a change in Body Mass Index (ΔBMI).
3. Subjects who are high in dietary restraint will become disinhibited and will show an increase in body weight, whilst those who are low in dietary restraint will increase in dietary restraint and show a decrease in body weight.
4. Subjects who are low in mastery will increase their bingeing behaviour.
5. Subjects who increase their bingeing behaviour will show increased consumption of foods that are high in carbohydrate and saturated fats.

1.2) Sociocultural influences on body weight and obesity.

Interest in dieting and its effects on body weight has increased considerably over the past three decades. This is not surprising given that there is evidence of obesity epidemic in modern Western environments (Rennie and Jebb 2005;Valdez and Williamson 2002). Specific factors such as a) the stable and abundant food supply, b) the wide availability of processed, high fat, high carbohydrate and palatable foods, c) large portion sizes, and d) the prevalence of sedentary life styles all contribute to the striking increase in obesity rates (Nielsen et al 2002). In the UK, recent figures show that 23% of men and 25% of women were obese in 2002 (Rennie and Jebb 2005), in the US obesity now stands at 31% (OECD 2004). In response to this it is suggested that Western society has become increasingly obesogenic (Slof et al 2003).

The notion that sociocultural influences promote disturbances of body image and eating patterns has become a prominent contemporary issue with the concurrent increase in obesity in developed countries. These sociocultural influences primarily emanate from the mass media, from family and from peers. They focus on the idealization of thinness, physical fitness, and the disparagement of being overweight (Stice 2000). These influences support and perpetuate the thin-ideal body image for most women. Social reinforcement and modelling are proposed as two mechanisms promoting attitudes and behaviours that result in a thin-ideal body image and unhealthy weight control behaviours, such as excessive dieting and binge eating (Thompson et al 1999). It is suggested that these attitudes and behaviours may result in dieting and negative affect, thereby increasing the possibility of unhealthy dieting or other weight control behaviours and eating pathology. However, this is not the case for all women: a recent twin study has shown that women who are persistently thin

display lower rates of dieting and binge eating, have higher self esteem and lower perfectionism and body dissatisfaction (Slof et al 2003). Despite sociocultural pressures, these women remain thin. It is recognized that there is likely to be a genetic locus for thinness in these individuals (see the next section, for a discussion on this topic). Following on from the discussion above, the variables, *stress*, *dietary restraint*, *bingeing behaviour* and *mastery* have been examined in the present study in an attempt to identify any correlation with body weight changes.

Obese people are often stigmatised in part because there are dichotomous views as to the causes of obesity (Friedman 2000). One view suggests that obesity arises due to a fundamental lack of discipline in life-style behaviours such as those related to diet and exercise. The diet industry supports this view to promote its financial interests. An alternative view suggests that body weight is physiologically controlled and that any alteration in weight either positive or negative elicits a powerful counter regulatory response to resist this change. The methodology utilised in this study clearly supports the later view in that individual differences play a vital role in the control of body weight, within the context of a psychobiological system.

1.2.a) Body weight, genes and obesity.

Body weight and height are measured as BMI, and is a measure of weight (kg) / height (m^2), and therefore does not differentiate between muscle and fat, or any subsequent

changes in these tissue. The underlying assumption therefore is that most variation in weight is due to changes in fat mass.

There are differences between countries in BMI values, probably mostly due to socio-economical and genetic influences. For example, China has a low mean population BMI whilst the USA has a high average population BMI. Age is also a factor with BMI increasing with age for both men and women until the age of 64 (Ogden 2003; Andres et al1985). The range of normal BMI is currently accepted internationally as a BMI of 18.5-24.9 kg/m^2 .The World Health Organisation (1998), classifies a BMI $\geq$ 25 kg/m^2 as overweight, for an adult and a BMI $\geq$ 30 kg/m^2 as obese and a BMI $\leq$ 17.5 kg/m^2 as underweight.

Genetic studies are beginning to unravel the complex nature of the phenotype, weight, and its reflection of an individual's genotype. For example, it is unknown how many overweight individuals have developed their condition as a result of a major deficiency in one gene (Bouchard, 2002), or a polygenic focus.

The psychobiological appetite and satiety system is a complex control system for weight control. With respect to the genetic component, body weight and obesity reflect the presence of a complex genotype rather than a more simple Mendelian type relationship in which there is generally a one to one association between genotype at a single locus and the presence or absence of a particular allele (Comuzzie and Allison 1998). Animal studies have provided evidence that genetics play an important role in the regulation of body weight. Environmental variables however confound identifying such loci in human obesity. Nonetheless, human studies indicate that genetic factors

account for a substantial proportion of variation in human adiposity (Barsh, Farooqi and O'rahilly 2000; Bouchard et al 1990).

As adaptation is vital to survival, there must be an interaction between the genotype and the environment. Inter-individual responses also, vary markedly to a number of dietary regimes and exercise programmes. Hunter gatherers lived in times of privation and the availability of food was extremely variable. These conditions were favourable to individuals whose genes predisposed to obesity and an increase in energy stores. Thus conferring an advantage and increasing the likelihood of survival in times of famine. James Neel, (1962; 1982), proposed this scenario as "the thrifty gene hypothesis". The magnitude of the change in phenotype (BMI), in response to environmental change, and whether these differences are genotype dependent, has yet to be examined. In the USA what does seem to be emerging from population trends in BMI, is that the lean possess genes which appear to protect the individual from obesity (Friedman, 2003). The obese however appear to possess a genetic susceptibility to an increase in body weight in the presence of an abundant food supply.

Animal studies indicate that body weight is controlled by many different genetic loci (Falconer and Mackay 1995). The A^{y}, *db*, *fat*, *ob* and *tub*, mutations have been recognised as spontaneous variants, of modern inbred strains of mice. Recent advances in genetic engineering and cloning techniques have provided considerable insight into the biology that underlies each mutation (Bulik et al 2003: Chagnon and Bouchard 1996: Leibel et al 1997). These techniques are important to our understanding of human body weight regulation and may provide a potential mechanism in the treatment of body weight disturbances.

What is the genetic contribution to BMI? Studies utilizing identical twin and fraternal twins, or identical twins reared apart suggest that heritability of BMI is in the region of 70%. Adoption studies estimate 30%, whilst family studies indicate around 50% (Maes, Neale and Eaves 1997).

What is the genetic contribution to obesity? The prevalence of obesity is twice as high in families of obese individuals, than in the population at large. The risk increases with the severity of obesity in the proband. It is estimated that the risk of extreme obesity (BMI ≥ 45 kg/m^2), is approximately seven to eight times higher in families of extremely obese individuals. This information was estimated using the lambda value (*This is the ratio of the risk of being obese when a biological relative is obese compared to the risk in the general population*), from 2,349 first-degree relatives of 840 obese probands and 5,851 participants of The National Health and Nutrition Examination Survey III 1998-1994 in the USA. The transmission of risk of obesity does not appear to be sex-specific, as studies to date have not found that obese parents differ in the frequency of having obese boys or girls.

Determining a genetic influence is confounded by multiple variables. It would appear that a genetic aetiology for human obesity is difficult to reconcile when the marked variation in prevalence observed as a function of socioeconomic and demographic variables are accounted for (Hill and Peters 1998; WHO 1998).

Many genes have been identified that have a contributory role i.e. a small effect, a few can have a major effect.

1.3) Stress and eating behaviour.

The possible involvement of stress in the development or maintenance of altered eating patterns has been a focus for investigation for some time (Connan et al 2003; Epel et al 2001; Oliver and Wardle 1998; Janzen, Kelly and Saklofske 1992). Stress is an important factor in the development of eating disorders such as anorexia nervosa, and bulimia nervosa (Cattanach and Rodin 1988). Understanding predictors of stress induced changes in eating behaviour is important, as stress can trigger relapses into both obesity (Wadden and Stunkard 2002; Rand and Stunkard, 1978), and bulimic episodes (Lingswiler et al 1989). Individual physical responses to stress may help to explain why some people tend to eat while others lose their appetite at times of stress (Wallis and Hetherington 2004; Dallman et al 2003; Epel et al 2001). Psychological literature supports the notion of a relationship between stress and eating behaviour, embodied in concepts such as *comfort food* and *comfort eating*. Stress has been linked to both an increase in eating (ie a hyperphagic response) and a decrease in eating (ie a hypophagic) response (Cartwright et al 2003).

Stressors may be physical, psychological or psychosocial in nature. Physical stressors include environmental pollutants, extremes of temperature, exercise or injuries or other trauma to the body. Psychological stress results from reactivity to one's own personal thoughts and/ or feelings about real or imagined threats. Changes in cortisol secretion and in mood are well known to occur during responses to stress (Brown et al 2004; Wardle et al 2001). These variables are also known to be modulators of stress induced eating. Epel (2001), in a controlled laboratory study, using female volunteers

found individuals who secreted high levels of cortisol consumed more calories, and sweet foods, in a stress condition compared to those who secreted lower levels and a control group. Increases in negative mood in response to stressors were also significantly related to greater food consumption. The present study explored this psychophysiological response in terms of food preference, cortisol reactivity and changes in body weight. Over time, these alterations may impact on both BMI and health.

1.3.a) The physiology of stress.

Cannon (1911), Cannon and Paz, (1914), were the first to describe the "fight or flight" response, which at that time focussed mainly on the neuroendocrine response and the release of adrenaline from the adrenal medulla. In the 1930's Hans Selye, one of the founders of the study of stress, studied the effects of glucocorticoids on target tissues. He advanced the "fight or flight" theory, and coined the phrase General Adaptation Syndrome (GAS), (Selye, 1951). Adrenaline remained important in the GAS, but the importance of glucocorticoids in resilience to stress was seen for the first time as a key feature. From the 1950's to the present time, researchers from various scientific disciplines have investigated the role of glucocorticoids on physiological system changes during the stress response and homeostasis. For example, cardiovascular functioning, fluid volume and haemorrhaging, metabolism, immunity and neurobiological effects. (see Saplosky et al 2000).

1.3.a.1) Glucocorticoids and the stress response.

Glucocorticoids have a major effect on food intake and a variety of stressors are capable of producing stress-induced changes in glucocorticoid secretion and eating behaviour in animal and human studies.

Corticotropic Releasing Hormone (CRH) containing neurones and CRH receptors are present in the brain and are particularly dense in the hypothalamus, pituitary, limbic system, prefrontal and cingulate cortices and autonomic structures (Chalmers et al 1995; Steckler and Holsboer 1999). CRH secreting neurones such as those in the hypothalamus play a central role in the release of adrenocorticotrophic hormone (ACTH) from the anterior pituitary and therefore cortisol from the adrenal cortex. CRH projections are therefore in significant locations to mediate emotional, behavioural and physiological responses to stress including eating behaviour. CRH is anorexigenic and in response to chronic stress this would be expected to result in weight loss (Dallman 2004; Connan 2003). As part of the normal adaptive response to chronic stress, the control of ACTH release becomes increasingly dependent on central organic vasopressin (AVP). This helps to prevent excessive weight loss, as a consequence of chronic stress. A failure in this mechanism may be a factor in the genesis of anorexia nervosa (Connan 2003).

Eating behaviour is of course complex and is associated with higher brain centres and cognition. The bidirectional pathways that connect higher brain centres to endocrine control systems in the hypothalamus are unclear, but for example, 5HT systems may be involved. 5-HT has hypophagic effects, which are more pronounced in female than in male animals (Ward *et al* 1998). Arousal and stress in rats increases central 5-HT

release (Dunn and Welch, 1991). However, activation of corticotropin-releasing hormone (CRH) is viewed as the final common neurobiological pathway responsible for producing stress-induced anorexia (Morley and Blundell, 1988). Centrally administered CRH is a potent reducer of nocturnal and starvation induced feeding and the hyperphagic effects produced by neuropeptide Y (NPY) are antagonized by CRH (Heinrichs et al 1993). The effects of CRH on feeding have been localized to the paraventricular nucleus (PVN), of the hypothalamus where noradrenaline acts to increase feeding and 5HT to decrease feeding. There is evidence that 5HT modulates the release of CRH at the hypothalamic level (Compaan et al 1996).

1.3.a.2) The central role of cortisol in eating behaviour.

Cortisol has an important role in energy regulation, for example by increasing available energy through gluconeogenesis and lipolysis (Cherrington, 1999). In animals prone to obesity (either genetically or as a result of brain lesions), glucocorticoids cause hyperphagia and weight gain (Bray, 1985). In rats, adrenalectomy and glucocorticoid receptor antagonists prevent or reverse obesity (Okada et al 1992), whereas administering corticosterone leads to an increased appetite for sucrose (Dallman et al 2004; Bell et al 2000), to hyperphagia and to weight gain (Flatt, 1989).

In humans, stress induced increases in plasma cortisol may have a role in the genesis of obesity. For example in patients with cancer, prednisolone (a cortisol receptor agonist), significantly increases appetite, compared to a control group (Willox et al 1984). In healthy men, administering cortisol for four days slightly increases energy expenditure but causes a dramatic increase in food intake (Tataranni et al 1996). It is

therefore predicted *in this study*, that endogenous increased secretion of cortisol due to stress, will play a significant part in stress induced eating and weight gain, and that *high cortisol secretors will increase in weight significantly more than low cortisol secretors*.

Macronutrient selection may also be altered by stress for example, women tend to prefer high fat or sweet foods when moderately stressed (Grunberg and Straub, 1992; Klein et al 1996). There is also evidence that adrenal steroids influence macronutrient selection, by increasing appetite primarily for carbohydrates, and also for fat, and regulating the timing of eating in rodents (Dallman et al 2003; McEwen et al 1993; Tempel and Leibowitz, 1994). The effects of stress on restrained and unrestrained eaters have been examined by employing psychological and physical stressors. *In this study, it is predicted that those who secrete more cortisol in response to stress will tend to eat more calories, as well as choose sweet or high fat foods. It is also expected that restrained eaters will increase their intake of food, and switch to a higher carbohydrate and fat content, whilst unrestrained eaters will not.*

Psychological factors are the most frequently reported stress stimuli, and are reported as the reason for most stress responses by individuals (Levine and Ursin 1991). The effects obviously depend on the individual i.e. on their previous stimuli or learning. Emotional arousal has been associated with both increased or decreased food intake and weight (Stone and Brownell 1994; Willenbring et al 1986), but little is known about the mechanisms that determine the direction of change. Clearly, understanding predictors of stress induced eating is important, as stress can trigger relapses into

bulimic episodes (Lingswiler et al 1989) and to obesity (Dallman et al 2004; Rand and Stunkard 1978), and in general, may be a significant contributor to problematic eating.

Studies of the association between stress and eating have developed in several different disciplines (Wardle et al 2000). Animal studies have investigated eating behaviour as part of the overall psychobiological reaction to stress (Robins and Fray 1980, Ely et al 1997), while human studies of eating disorders usually consider stress as a factor that disturbs food intake regulation, body weight and restraint (Polivy 1994). In humans, there is limited research, identifying a relationship between eating behaviour and stress. Cross sectional studies attempting to identify an association include laboratory stressors (e.g. viewing a stressful film Grunberg and Straub (1992), preparing for public speaking), and cross sectional cohort studies (work stress) (Wardle 2000), and academic examination stress (Macht et al 2005;Weidner et al 1996; Pollard et al 1995). These studies provide cross sectional accounts of amounts and types of food eaten, using no controls to reduce the effects of confounding variables of time, stress and food availability. Subjects who tend to eat in response to laboratory stressors may simply eat less at the next meal to regulate energy intake. The next section explores research approaches and studies investigating the relationship between stressors and eating behaviour in more detail.

1.3.b) Stress, eating behaviour, and research approaches.

Stress and eating behaviour have been explored using several different research approaches. A great deal of research, exploring the interaction between stress and eating behaviour is based in a laboratory or clinic setting. Choice of food, such as fat

or sugar content (Dallman et al 2003), has often been considered a responsive behaviour to life stress either inadvertently or as a deliberate strategy for coping with stress (Lattimore and Caswell 2004; Folkman & Lazarus 1980). This responsive behaviour could arise from a general effect of stress on food intake, for example through physiological changes. Alternatively, changes in eating behaviour may arise from significant individual differences in responses to stress, such as dietary restraint or alteration in mood. Stress is widely reported to lead to overeating in some individuals (Yacono, Freeman and Gil 2004), and a consistent finding from studies measuring these individual differences is that those scoring high in dietary restraint eat more under stress, whereas intake is the same or lower in unrestrained eaters (Lattimore and Caswell 2004).

Animal studies have investigated eating behaviour as part of the overall psychobiological response to stress (Dallman et al 2004; Ely et al, 1997). These studies have produced evidence of both hyperphagia and hypophagia in response to stress (Dess et al 1998; York, 1992; Robbins & Fray, 1980). Whilst these animal studies have been instrumental in investigating physiological mechanisms underlying eating behaviour, psychological mechanisms driving this behaviour are largely inferred. Many animal and human studies focus on the amount and type of food consumed following a stressor. The findings therefore relate to cross sectional, rather than longitudinal, variation in eating behaviour. More attention needs to be given to naturalistic, long term effects, particularly individual differences which impact on changes in weight. *In this study individual differences in stress response (cortisol secretion), mastery, mood, dietary restraint, bingeing behaviour, and food choices (in a sub group), will be explored.*

Interest in dieting, overeating, and stress has increased during the past three decades. Eating behaviour of normal weight and overweight individuals in early studies focused on weight or BMI, as a defining variable. For example, overweight people were observed to increase food intake during stressful time periods. More recent research focuses on restraint theory and eating behaviour, rather than weight status alone. Eating behaviour of restrained eaters has been shown to differ in important aspects from unrestrained eaters. For example, the earliest studies of dieting, using experimental starvation in conscientious objectors found periods of food restriction were frequently followed by bouts of overeating or frank bingeing (Keys et al 1950). This finding, coupled with more recent observations that unrestrained eaters, following a high calorie meal will compensate by eating less calories in the ensuing time period (Laessle et al, 1996), suggests that eating behaviour and weight do not have a simple linear relationship. In other words, the association between unrestrained and restrained eating behaviour, and weight, is not a simple linear model, and restrained eating behaviour is multifactorial. For example, some restrained eaters become unrestrained in response to stress, or plan to eat a diet breaking meal later in the day, which increases consumption "The Counterregulation Theory" (Herman & Polivy, 1984), whilst others maintain restraint or increase restraint behaviour (Westenhoefer et al 1999). It would seem therefore that stress might result in either decreased or increased food intake in susceptible individuals. This dichotomy is referred to as the "stress eating paradox" (Stone & Brownell 1994). Research design and methodology has been developed from animal and human studies, with animal studies focussing mainly on observable eating behaviour and underlying physiological changes and, human, clinical and laboratory studies on psychological aspects of eating behaviour such as

restraint, anxiety and depression. Greeno and Wing (1994), in a comprehensive review of the literature on the relationship between stress and eating behaviour, attempted to provide a contemporary framework of research design and methods utilised in this research. They proposed two models to explain this paradox, namely "the general effect model" and "the individual difference model".

The general effect model predicts that stress changes food intake generally, most likely through physiological adaptation, whilst the individual difference model predicts stress only causes changes in the eating behaviour of vulnerable individuals. Accordingly, the individual difference model incorporates complex, psychological antecedents into the design and methodology, utilised in these studies. The following overview of the general effect and individual difference model, provides part of the basis for the present studies design and methodology.

1.3.b.1) The general effect model

The general effect model lends itself to physiological explanations of stress induced eating. Most research utilizing the general effect model has been directed toward

finding physiological mechanisms, to explain stress induced eating in a laboratory setting. This model has been tested primarily in animals, and in contrast to the individual difference model, does not include psychological factors involved in eating behaviour. The advantage of laboratory stressors is that each subject is exposed to the same stressor. However, these stressors are weaker and shorter than are naturalistic stressors (Greeno & Wing 1994). For example, the tail pinch procedure which is one of the principal methods used as an acute stressor, for testing stress induced eating in rodents, increases oral behaviours, including eating (Nemeroff et al,1978; Antelman et al, 1975). Despite the absence of a control group, these studies formed the basis for later pharmacological studies, which utilised pharmacologic agents that increase or decrease tail-pinch-induced eating (Antelman, Eichler, Black, & Kocan, 1980; Junquera, Lanzagorta, & Russek, 1987; Nobrega, Dixon, Troncone, & Barros, 1989). These studies have been used to identify CNS systems and substances such as endogenous opiates (Morley & Levine 1980), that are involved in stress induced eating.

The design and methodology utilised in these studies somewhat weakens support for their findings, namely, that tail pinch will induce eating as a result of stress. Many of the studies have used small numbers of animals. Testing conditions also varied in that, for example, tail pinching varied between studies from one to several pinches per day, each lasting from a few seconds to a few minutes. The pinched animals were usually tested outside of their regular living quarters, whilst the control animals remained in their living quarters. Coupled with this, it is also unclear that animals find the tail

pinch procedure stressful, and therefore it is questionable that tail pinch responses really provide an example of stress induced eating.

Electric shock is also used as an acute stressor in rodent studies and effects on eating behaviour, and provides inconsistent results. It has been shown to increase overall eating (Siegal & Brantley 1951; Ullman, 1951), to decrease overall eating (Sterritt, 1962; Sterritt & Shemberg, 1963; Tugendhat, 1960), and not to affect eating (Sterritt, 1965). One study reported different effects for different types of food (Strongman, 1965). Recently, Hagan, et al (2003) developed a new model of stress induced binge eating. In their model, they showed that following a history of food restriction, electric shock elicited transient large increases in overeating if rats were offered even a taste of palatable food. In two separate experiments using different rats, the effect did not occur until after a minimum of three restriction stress cycles, suggesting neuroadaptive processes may induce the development of binge eating. Short term physical stressors such as a cold swim have also been found to increase eating in animals (Vaswani, Tejwani, & Mousa 1983). However, these stressors, and the short term increased eating behaviour, have not been tested longitudinally for any impact on body weight.

Socialisation and housing conditions particularly isolation, are the only consistent stressor models in animals to cause an increase in eating and body weight in rats. Data from animal models, utilizing rat pups, have demonstrated that early life experiences play a crucial role in the maturation of biological systems of the brain (Francis & Meaney 1999), and thus may impact on eating behaviour and body weight. By the time of adolescence, maternally deprived rats weigh significantly less than their

nondeprived counterparts (McIntosh et al 1999). In the rat and mouse, postnatal handling decreases the magnitude of behavioural and endocrine responses to stress in adulthood (Meaney et al 1996). These effects persist throughout the life of the animal (Meaney 1998), and are associated with differences in health outcomes under conditions of stress. The central Corticotrophin Releasing Factor systems are critical targets for these environmental effects (McEwan 1998), with eating behaviour being of major concern in the present study.

In a laboratory study which involved inducing stress with a stressful film (Grunberg & Straub 1992), it was found that men's intake of food was lower in the stress than the control condition and women's intake did not change. The food offered was a snack and therefore the total intake of food in this study was very small. In another study, midday meal intake on the day before a surgical operation was compared with the midday meal intake a few weeks later (Bellisle et al 1990). No difference in amount of food, energy intake or dietary composition was found. The small number of participants n = 12, gave this study limited statistical power. These variations in results could also be due to individual differences in stress induced eating, such as restraint. These findings and the limitations of the general effect model emphasise the need for the inclusion of psychological antecedents, and the value of the individual difference model.

Findings from the general effect model have enhanced knowledge related to stress and the physiological basis of eating behaviour. However, eating behaviour would seem to be a reflection of a more complex interplay between physiology, eating behaviour and psychological antecedents used in an adaptive response to stress. "Human –animal"

dualism (de Waal 2002), suggests that humans possess some psychological capacities that animals do not, and that none of our psychological processes are shared. Whilst the general effect model relies heavily on animal models, there can be little doubt that it has contributed to our understanding of human eating behaviour.

1.3.b.2) Individual Difference Models, stress and dietary restraint.

The second model is the individual differences model, which has been used extensively in human studies, and posits that individual differences in learning history, attitudes, or biology, determine the effects of stress on eating. Early research suggests two ways of identifying individuals who will be predisposed to stress induced eating. Firstly, normal weight individuals decrease their eating while stressed, whereas eating by obese individuals is unaffected by stress (Schachter, Goldman, & Gordan, 1968). Secondly, individuals who endeavour to control their eating (restrained individuals), increase their eating while stressed, whereas those who do not have to work to control their eating (unrestrained eaters), are unaffected by stress (Herman & Polivy, 1975). Further discussion on dietary restraint can be found in 1.7.

Research into causative factors of obesity triggered the development of the individual difference models. Stunkard, (1959), in clinical observations, proposed the hypothesis that overweight people were more likely to report eating under stress than people of normal weight, and that normal weight and obese people had different eating

characteristics in response to stress. From this research, the individual difference model developed with the view that eating behaviour resulted from a complex interaction of not only biological determinants (as the general effect model would suggest), but also of complex interaction between learnt behaviour and psychological antecedents. Early studies using individual difference models showed that only some overweight people respond to stress by eating more (Baucom & Aiken, 1981; Schlundt et al., 1991; Van Strien et al., 1986). One study (Baukom & Aiken, 1981), suggested that dieting, not obesity, is the relevant predictor of stress induced eating. Baukom & Aiken (1981), selected dieting and non-dieting , obese and normal weight subjects and found that dieting, and not weight category, predicted response to stress. Stressed dieters ate more than dieters who were unstressed, regardless of weight category. However, their study induced a depressed or non depressed mood in obese and non obese individuals. Introducing the factor of depression into their study may have further confounded the relationship between dieting and stress related eating behaviour. The failure to control for restriction and weight could also contribute to this pattern of contradictory findings. Therefore, the present study incorporates these important variables in both dietary restrictors and non-restrictors.

Laboratory studies, utilizing the individual difference model, have been used extensively to measure food intake in female college students during periods of high stress and low stress. Dietary restraint and food consumption have been monitored in response to a stressor. Consistent observations of hyperphagia in women who are restrained eaters, and either hypophagia or no change in food intake, in women who are non-restrainers, have been reported (Herman et al 1987; Schotte et al 1990; Heatherton et al 1991; Wallis and Hetherington 2004). These laboratory studies induce

negative affect, for example by viewing a distressing film, or subjecting individuals to a physical or ego threat. Conversely, these findings are challenged by a laboratory study, which reported no impact on dietary restraint on stress induced eating, following anticipation of public speaking (Oliver et al 2000). How realistic these stressors are in a natural environment is debatable. Therefore, there is a need for a shift towards a naturalistic, longitudinal approach to stress and eating behaviour as utilised in the design of this study.

Several naturalistic studies comparing periods of high and low life stress, with energy intake and food choice, have revealed mixed outcomes. McCann et al (1990), used occupational stress and variation of work load on female office workers and found a higher energy intake during times of high workload conditions compared to low work load conditions. Michaud et al (1990), used examination stress in high school students and found a higher food intake during the twenty-four hour period on the day of an examination, compared to intake one month later. Weidner et al (1996) found with undergraduate students that nutritional choice, as a health behaviour, deteriorated under stress. For example, there was a tendency toward an increase in sugary, fatty, snack type foods. No control groups were used in these studies and high and low stress periods were a feature rather than a longitudinal element in the design.

More recently, Yacono-Freeman and Gil (2004), evaluated whether psychological stress, use of specific coping strategies, and dietary restraint would prospectively predict binge eating episodes. Forty-six binge eating college women kept diaries assessing affect, stress, coping and binge eating for 30 days; restraint was measured at baseline. They found that women with high dietary restraint showed different patterns

of relationship for stress, coping and binge eating, than women with low dietary restraint. Women with low or average (but not high), restraint were associated with greater increases in the likelihood of same day binge eating as psychological stress increased. The nature of self report (in terms of retrospective recall), coupled with a small sample size and difficulty in collecting some of the diaries must be borne in mind when considering the reliability of this study. Wardle et al (2000), examined the association between work stress and nutritional status in relation to dietary restraint in a community sample of adults. The design included a cross sectional and a longitudinal element. They reported a significant moderating effect of restrained eating, with a hyperphagic response to work stress in restrained eaters, compared to no effect in unrestrained eaters. These are interesting findings from a naturalistic study. The results indicate that the associations between restraint and stress induced eating that have been observed in the laboratory extend to the natural environment.

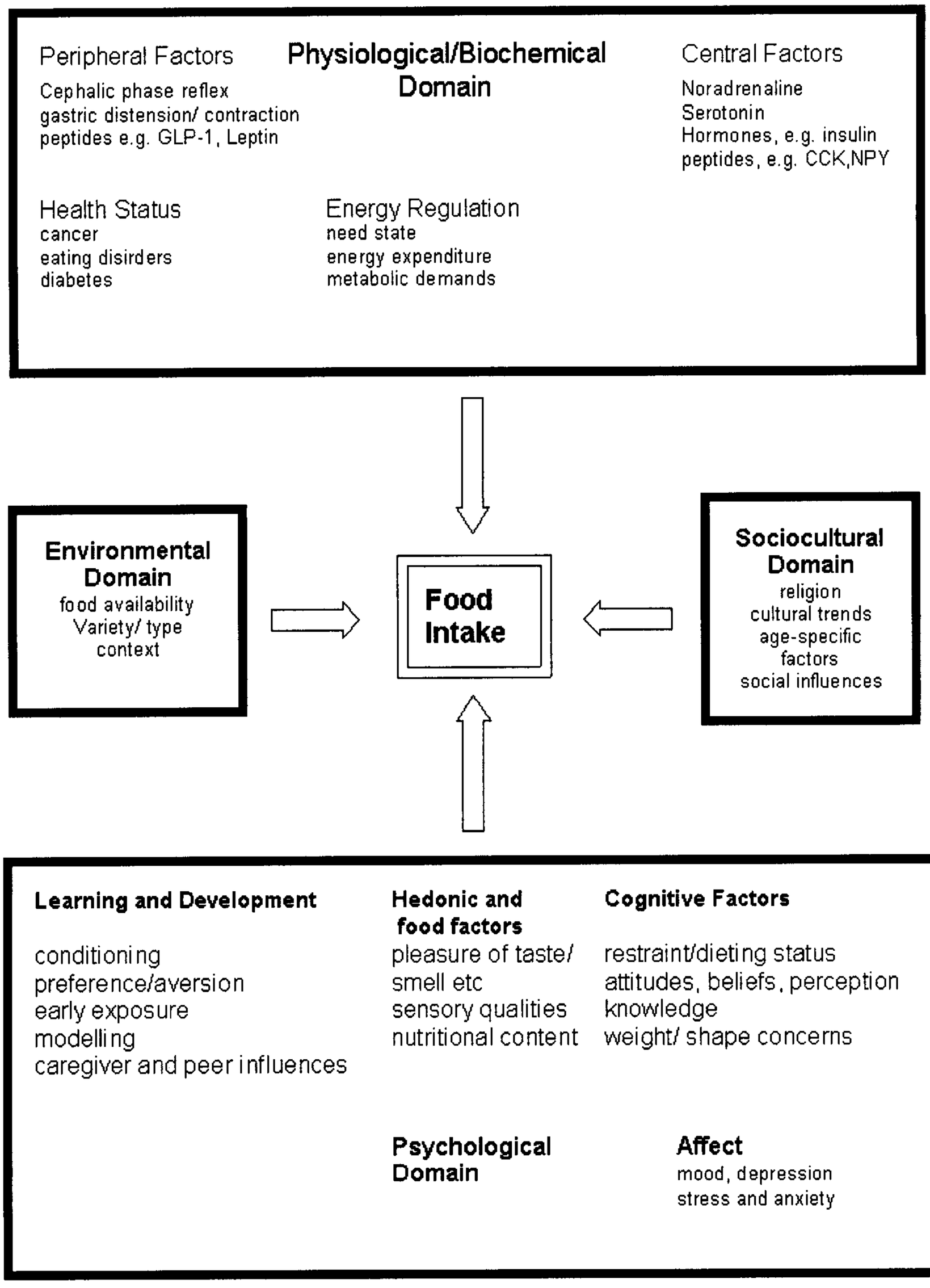
1.3.b.3) In summary

The general effect model and individual difference model as proposed by Greeno and Wing (1994), has been used as a mechanism, through which research exploring the relationship between stress, eating behaviour and weight can be evaluated. To minimise the criticisms of these research approaches, the present study has a naturalistic, cross sectional, longitudinal design and explores the relationship between stress, dietary restraint, and body weight.

1.4) Models of appetite regulation.

Hetherington (1993), categorised factors related to the regulation of food intake into four domains: These are the physiological/ biochemical, environmental, sociocultural and psychological *(Fig 1.3, page* 45*)*. Blundell (2000), presents a psychobiological system approach to appetite and weight control. In this approach, the psychobiological system permits an understanding of the interrelationships among behavioural events that comprise eating, peripheral physiology and metabolism, and central neurochemical processes. It is suggested that disturbances in any of these three levels of operation i.e. eating behaviour, peripheral physiology and metabolites, and brain activity, would lead to disruption in the expression of appetite (Blundell and Cooling 2000). For example, it is suggested that dieting is a form of behavioural control that desynchronises the appetite system, and that dieting may be reinforced by sociocultural influences.

Fig 1.3 Domains and factors regulating feeding and food intake.



Taken from Hetherington (1993) with minor adaptations. These factors operate as a whole to regulate feeding and food intake.

The purpose of this study is to investigate possible changes in human body weight, resulting from exposure to a naturalistic stressor, in the form of an academic examination using a psychobiological systems approach (Fig 1.4, page 58, appx 1 & 2, (Levine and Ursin (1991). and Lazurus and Folkman(1984), transactional approach will apply measurement methods to these levels of operation). (This study measured ***eating behaviour****(EDE Q4, and food choice FFQ),* ***peripheral physiology and metabolites*** *(cortisol),* ***mental health*** *(anxiety & depression),* ***mastery and coping****.*

1.4.a) Regulation of appetite, energy balance and body weight.

The regulation of energy homeostasis is controlled by a complex neuroendocrine system consisting of central and peripheral components.

Appetite, energy balance and body weight gain are modulated by a range of neurochemical, neuroendocrine and neural signals from diverse regions in the brain and different organs in the body. The hypothalamus plays an important integrative function in the process, acting through a variety of systems that involve an interaction between nutrients, amines, neuropeptides and hormones. These systems underlie normal nutrient intake and metabolism and are thought to be responsible for shifts in feeding behaviour across the circadian cycle and fluctuations relating to gender and age in both rodents and humans. Moreover, alterations in these normal neurochemical-neuroendocrine systems may be associated with abnormal eating patterns, such as anorexia nervosa, bulimia and obesity. With the rising prevalence of obesity and other eating disorders, a better understanding of the mechanisms involved in the regulation

of food intake and body weight is required. Understanding the systems that control eating behaviour might provide a foundation for the treatment and possible prevention of such disorders (Leibowitz, 1992). For a review of the literature on studies of this subject please see Hillebrand et al (2002); Kalra et al 1999; & Morley 1987, for reviews).

1.4.a.1) Control mechanisms involved in short term satiety.

The central and peripheral systems which interact to regulate satiety are summarised below.

Short term satiety is controlled centrally via the central nervous system (CNS), and is referred to as central control, whilst peripheral control occurs in the GI tract. It is of note that the GI tract contains the most complex neural system found outside of the CNS and therefore it is not surprising that there is a complex array of communication between the CNS and the gut and vice versa.

1.4.a.2) The hypothalamus and central control processes regulating satiety.

The hypothalamus is the principal centre in the CNS involved in the control of food intake. In the early 20th century, the hypothalamus was thought to play an important role in feeding behaviour. Experiments in the early 40's led to the Dual Centre Model, for regulation of feeding, in which nuclei in the lateral hypothalamus (LH), served as the feeding centre and the ventral medial hypothalamus (VMH), as the satiety centre

(Hetherington, 1940). Although this model has been questioned, the VMH and the LH, are now established as being involved in food intake (Bray and York, 1979), and include the arcuate nucleus (ARC), the paraventricular nucleus (PVN), and the dorsomedial nucleus (DMN). ARC neurons are situated around the third ventricle at the base of the hypothalamus and are referred to as *first order neurons* because of their direct contact with peripheral satiety factors such as insulin and leptin. The neurovascular anatomy in this region of the brain is such that in the median eminence (ME), which overlies the ARC, the blood brain barrier (BBB), is absent and the ARC terminal axons are in direct contact with the systemic circulation (Peruzzo et al 2000). The cell bodies of these axons however, are not in direct contact with the systemic blood stream as they are protected by the BBB. The ARC contains at least two distinct systems involved in the regulation of energy balance; for example, some neurons containing agouti-gene-related protein (AgRP), and neuropeptide Y (NPY), both of which are orexigenic neuropeptides; the anorexigenic neuropeptides, pro-opiomelanocortin (POMC), and cocaine-and amphetamine-regulated transcript (CART), are in other neurones (Hillebrand et al 2002). Neurons project from the ARC to *second order neurons* in the PVN, VMH and LH (Schwartz et al 2000). These second order neurons project to a number of areas including the nucleus of the solitary tract (NTS) in the brainstem and the dorso motor nucleus of the vagus (DMV). Communication in this system between the hypothalamic pathways and the caudal brainstem, responds to meal related satiety signals and is essential for the long term regulation of energy homeostasis.

1.4.a.3) Neurotransmitters and peptides

Several neurotransmitter and peptide systems are involved in the control of feeding. They are classified below according to their effects on feeding.

1.4.a.4) Orexigenic and anorexigenic neurotransmitters and neuropeptides

Neurotransmitters and neuropeptides act through specific receptors to regulate food intake. The signals are grouped into hunger inducing orexigenic stimuli and satiety inducing anorexigenic stimuli. Among the neuropeptides that activate hunger signals are noradrenaline (NA), neuropeptide Y (NPY), agouti related peptide (AGRP), melanin concentrating hormone (MCH), orexin A and B, and galanin. (Ritter et al 2003; Berglund et al, 2003; Hahn et al 1998).

Those which induce satiety include, corticotropin releasing hormone (CRH), serotonin (5-HT), leptin, arginine vasopressin (AVP), and melanocyte stimulating hormone (α-MSH). Most of these neuropeptides act through metabotrophic receptors that are functionally coupled to adenylate cyclase by stimulatory G proteins (Gs), or inhibitory G protein (Gi). Binding of anorectic neuropeptides to their respective receptor subtypes increases cAMP production from cellular ATP via Gs protein and adenylate cyclase activation. In contrast, association of orexigenic neuropeptides appears to activate a Gi protein complex, which leads to the inhibition of cAMP synthesis. Thus, coupling of a neuropeptides receptor to either a Gs or Gi protein determines whether intracellular concentration of cAMP will increase or decrease. The negative correlation between the cAMP synthesis and food intake suggests that cAMP is an important

biochemical mediator in the regulation of feeding behaviour (Bannon et al 2000; Chalmers et al 1995; Steckler and Holsboer 1999).

The next section describes neurotransmitters and neuropeptides involved in the control of appetite during stressful periods.

1.4.a.5) Noradrenaline

Noradrenergic neurones project to many brain regions although their neurons all arise from the locus coeruleus (LC), in the brainstem. Nuclei in the medulla of the hindbrain, play a pivotal role in the central distribution of sensory signals from the internal environment. Noradrenergic projections to the hypothalamic PVN influence various secretory patterns of the hypothalamic-pituitary-adrenal-axis (HPA) and are essential for feeding (Ruggiero et al, 1985; Tucker et al, 1987; Ritter et al 2003). Evidence indicates that that both pre- and post-synaptic adrenergic receptors control CRH neuronal function and thus shape HPA activation patterns (Plotsky et al, 1989; Whitnall, 1993).

Early studies identifying adrenergic projections to the PVN involved lesions, fluorescence histochemistry and brain cannulation to investigate their involvement in feeding behaviour (Leibowitz, 1980). Adrenergic agonists, when administered directly into the hypothalamus of satiated rats, produce a vigorous feeding response. In the medial hypothalamus, the PVN was identified as the most potent site for initiating this feeding effect (Leibowitz, 1978). The effects of noradrenaline in the PVN are mediated by α_2 adrenergic receptors and these effects are increased by corticosterone

[the rodent equivalent of cortisol] (Roland et al, 1986). Activation of PVN receptors stimulates ingestion of carbohydrate rich foods and corticosterone interacts positively with noradrenaline to potentiate this ingestion (Tempel and Leibowitz, 1993; Leibowitz, 1988). During a stress response α_2 adrenergic stimulation of the PVN produces an increase in circulating levels of both corticosterone and glucose. This state involves adjustments in carbohydrate (CHO) ingestion as well as metabolism, which allow the animal to maintain energy reserves during stressful periods. (In the present study, a food preference questionnaire was included to detect any changes in food-type preferences, such as CHO and saturated fat content, at time 1 and time 2 (ie at times of different levels of stress).

1.4.a.6) Neuropeptide Y

Neuropeptide Y (NPY), is abundant in the brain: it is stored in synaptic vesicles often with "classical" neurotransmitters such as noradrenaline (Allen et al, 1983). It has 36 amino acids and is a member of the pancreatic polypeptide family. Using immuno-histochemical techniques it has been found in high concentrations in the hypothalamus and locus coeruleus (DiMaggio et al, 1985). Two other peptides in this pancreatic polypeptide family, share a similar structure (known as the PP-fold), these are, pancreatic polypeptide (PP), and peptide YY (PYY). There are four known human receptors for the PP-fold peptides, namely Y1, Y2, Y4 and Y5, each of them being able to bind at least two of the endogenous ligands (Berglund et al, 2003).

NPY expression has a daily rhythm, which may be regulated by the circadian clock in the suprachiasmatic nucleus (SCN), in the hypothalamus. NPY producing neurons in

the ARC are co localised with AgRP and galanin, and express the leptin Ob-R_b receptor (Hahn et al 1998). NPY projections run to the PVN, from the ARC, which is itself also innervated by NPY neurons arising from the brainstem. These NPY projections also run to the ventromedial nucleus of the hypothalamus (VMH), and dorsomedial nucleus of the hypothalamus (DMH). From there, the NPY fibres are connected to the nucleus of the solitary tract (NTS), and dorsal vagal complex.

The localisation of these neural circuits suggests a role for NPY in somatic, sensory and cognitive brain functions. Plasma levels of NPY are significant and particularly sensitive to stressors that can bring about very large increases in the circulating concentrations (Pernow et al, 1986). The adrenal glands are the most likely source of the plasma derived NPY, during stressful situations. Plasma concentrations of PYY and PP are less dynamic, with increases in plasma concentrations being observed after meals (Hazelwood 1993). It is thought that many of the peripheral actions of neuronally released circulating NPY are shared with PYY (Gehlert 1999).

NPY administration into the cerebral ventricles or directly into the PVN increases food intake, decreases energy expenditure, reduces sympathetic outflow to brown adipose tissue, and increases lipogenesis by stimulating the expression of lipogenic enzymes in white adipose tissue (Billington et al, 1991; Stanley et al, 1986). In the PVN, NPY also alters the release of insulin, corticosterone and glucagons (Leibowitz et al 1988; Motz & McDonald 1985). Hence, NPY promotes positive energy balance and increases fat storage. NPY is also associated with vascular systems, memory, mood, neuronal excitability and reproduction (Bergland et al, 2003).

In rats, the most NPY sensitive hypothalamic area is the perifornical region. In rodents, NPY significantly reduces the latency to feeding initiation and increases meal size without altering meal frequency (Leibowitz et al, 1991). In rats repeated doses of NPY result in a consistent increase in feeding and body weight over a 10 day period. The increase in body weight consists primarily of increased fat mass (Zarevski et al, 1993), with no tolerance exhibited to the effects of NPY (Stanley et al 1989). The chronic infusion also results in hyperinsulinaemia and insulin resistance (Stanley 1993). The effect of NPY on macronutrient selection is an increase in carbohydrate and fat consumption, with the increase in carbohydrate consumption being the most pronounced (Stanley et al, 1985). These findings suggest that elevated endogenous NPY levels contribute to obesity and diabetic states.

1.4.a.7) Corticotropic Releasing Hormone

Corticotropic Releasing Hormone (CRH) is a 41 amino acid peptide first isolated from the ovine hypothalamus and is identified with its initiating role in the hypothalamic pituitary adrenal (HPA) axis. CRH containing neurones and CRH receptors are present in the brain and are particularly dense in the hypothalamus, pituitary, limbic system, prefrontal and cingulate cortices and autonomic structures (Chalmers et al 1995; Steckler and Holsboer 1999). CRH cell bodies are mainly found in the PVN and in the central nucleus of the amygdala (Gray & Magnuson 1987; Sakanaka & Shibasaki 1986), from which projections go to the hypothalamus, brainstem, NTS, and locus coeruleus (LC), (Gray 1993). CRH secreting neurones such as those in the hypothalamus have a central role in the release of adrenocorticotrophic hormone

(ACTH) from the anterior pituitary and therefore cortisol from the adrenal cortex. CRH projections are therefore in significant locations to mediate emotional, behavioural and physiological responses to stress (including eating behaviour).

CRH effects are mediated by two G-protein coupled receptors (GPCRs), which are positively coupled to adenylate cyclase and differ in their distribution. The CRH2-receptor (CRH-2), exists as CRH2α, CRH2β, and CRH2γ (Kostich 1998), have low affinity for CRH (Lovenberg et al 1995a; Lovenberg et al 1995b), and is mainly in the limbic regions. The CRH1-receptor (CRH1-R), has higher affinity for CRH and is mainly found in the pituitary and the cerebral cortex. Both CRH-2 and CRH-1 receptors are found in the cerebellum, brainstem and hypothalamus (Chalmers et al 1995; Lovenberg et al 1995b; Primus et al 1997). A CRH binding protein is also involved in CRH signalling and is located in the CNS and periphery. CRH binding protein modulates CRH activity by binding CRH and thereby reducing its free concentration in plasma (Cortright et al 1997; Karolyi et al 1999).

CRH is a potent anorexigenic peptide, which acts downstream from leptin. Experimental administration of CRH into the PVN inhibits both night time and fasting induced feeding. In the well fed state; leptin increases CRH expression and neuronal activity, with the opposite effects seen in the fasted state (Ahima et al 1996; Bannon et al 2000).

CRH is anorexigenic and in response to chronic stress this would be expected to result in weight loss. However, as part of the normal adaptive response to chronic stress, the control of ACTH release becomes increasingly dependent on central organic

vasopressin (AVP). This helps to prevent excessive weight loss: a failure in this mechanism may be a contributing factor to anorexia nervosa (Connan et al 2003).

Eating behaviour involves higher brain centres involved with cognition. The bidirectional pathways that connect higher brain centres to sensory systems in the hypothalamus are unclear, but for example, 5-HT systems may be involved. 5-HT has hypophagic effects which are more pronounced in female than in male animals (Ward *et al* 1998), and arousal and stress in rats increases central 5-HT release (Dunn and Welch, 1991). CRH activation is seen as the final common neurobiological pathway responsible for producing stress-induced anorexia (Morley and Blundell, 1988). Centrally administered CRH is a potent reducer of nocturnal and starvation induced feeding. The hyperphagic effects produced by NPY are antagonized by CRH. The effects of CRH on feeding have been localized to the PVN where noradrenaline increases feeding and 5-HT is inhibitory. There is evidence that 5-HT releases CRH at the hypothalamic level. (see page 31, for the effects of cortisol secretion on metabolism and eating behaviour).

1.4.a.8) Leptin and Insulin

Leptin and its receptor (Ob-R), are part of a system that regulates energy balance through behavioural and metabolic effectors. The production and release of leptin by adipocytes serves as the afferent loop in the regulation of this energy balance. Leptin acts on the hypothalamus, as a tonic signal, the absence of which triggers a series of neuroendocrine responses responsible for the conservation of energy when food availability is limited. A decrease in body fat results in a reduction in circulating levels of leptin, which in turn stimulates food intake (Takahashi et al 2002; Ahima et al 1996). A total deficiency in or resistance to leptin causes severe obesity. As leptin

levels rise with increasing adiposity in rodents and man, it acts as a negative feedback 'adipostatic signal' to brain centres controlling energy homeostasis, limiting obesity in times of nutritional abundance. Intrinsic sensitivity to leptin is variable and this may be one factor which helps to explain why some individuals are obese and others are not (Friedman and Hallas, 1998; Maffei et al 1995).

The first hormone to be implicated in the control of body weight was insulin, a pancreatic hormone. Insulin enters the brain from the circulation and acts there to reduce energy intake (Woods et al 1979). As weight increases, insulin secretion increases in both the basal state and in response to meals to compensate for insulin resistance if normal glucose homeostasis is to be maintained (Kahn et al 1993; Polonsky et al 1998). Hyperglycaemia ensues if the pancreatic β cells fail to achieve this increase in secretion and most likely contributes to type-2 diabetes in obesity.

Leptin secretion from adipocytes and factors which control synthesis and secretion are different from that of insulin. A key factor linking leptin secretion to body fat mass is the rate of insulin stimulated glucose utilization in adipocytes (Mueller et al 1998). Leptin levels are lowered in both rodents and humans by food deprivation. The rate and time span of reduction is more rapid than would be expected from the decrease in body fat content. This is thought to be due to three factors during food deprivation. Firstly, a reduction in the release of insulin, secondly, a reduction in insulin stimulated glucose utilization in adipocytes and thirdly a reduction of glucose metabolism via the hexosamine pathway (Wang et al 1998). Leptin secretion may then be transiently dissociated from levels of total body fat. This exaggerated early decline of leptin levels would enable compensatory responses to be activated before energy stores are

substantially depleted (Schwartz et al 2000). In poorly controlled diabetes mellitus, low circulating levels of insulin and leptin result in diabetic hyperphagia. A rat model study established that by replenishing leptin (but not insulin), to non diabetic levels, diabetic hyperphagia was prevented (Sindelar et al 1999). The conclusion was that a deficiency of leptin, but not insulin, is required in this model, indicating that leptin has the more critical role in the control of energy homeostasis.

1.5) Life events, activation theory and mastery.

Several retrospective studies investigating altered eating behaviour (in bulimia and anorexia nervosa), have suggested an association between the onset of altered eating behaviour and major life changes (Lacey, Coker and Birtchnell, 1986; Pyle, Mitchell, and Eckert, 1981). Within non-clinical subjects (college women), positive correlations have been reported between severity of binge eating and the number of major life events experienced during the past month, and the amount of stress experienced in the past year (Hawkins and Clement, 1980), although Greenberg and Harvey (1986) did not report the same relationship. A longitudinal, prospective study of college students reported a link between the development of severe eating disturbances over the course of a school year and high perceived stress (Striegel-Moore, et al., 1989).

The somatic response to external and internal CNS stimulation is a widespread general response, affecting many bodily functions. There is specificity due to individual response profiles, either to the stimulation or to the individual. This general response is referred to as activation (Ursin, 1980). Ursin (1998), also presents a cognitive

approach to stress theory, with a simple model for the relationship between the psychological factors and the endocrine responses to stress.

1.6) Stress Process Models

A major problem with the concept of stress is its composite, multidimensional nature. Levine and Ursin (1991), identify three main subclasses (depicted in fig 1.4 below).

Fig 1.4. Schematic view of the transactional (Lazarus and Folkman 1984), and activation theory (Levine and Ursin 1991), combined.

	Associated factors in the present study
1. Input: Stress - Stimulus	(Stressor)*Examination*
2. Processing Systems + Subjective experience	(Psychological) *Mastery/coping/filters*
3. Output stress responses	(Neuroendocrine) *HPA axis/ cortisol*

See appendix 1 and 2, for a schematic view of the transactional (Lazarus and Folkman 1984), and activation theory (Levine and Ursin 1991), combined.

The conceptual strategy above forms a stress process framework which has been described by Skaff et al (1996). This framework views stress as a dynamic framework unfolding over time and involving three principal domains: stressors, resources and

outcomes. In essence, a similar process model to that presented above, except that processing systems are replaced by resources.

The present study explored the role of processing systems and subjective experience in the psychoneuroendocrine response to stress. The psychoneuroendocrine response to stress is also involved with the development of eating disorders. Numerous studies implicate changes in the (HPA) axis in clinical groups suffering from eating disorders for example, anorexia and bulimia nervosa (Connan et al 2003; Wellberg and Seckl 2001; Francis and Meaney 1999; Plotsky and Meaney 1993).

Recent attention has shifted toward investigation of variables, which may play a mediating role in the relationship between exposure to potentially stressful events and the effects of those events on the individual (Polivy and Herman 1998; Cattanach and Rodin, 1988). It has been suggested for example, that bulimic women may lack the necessary coping skills to deal effectively with difficult situations (Lacey, et al., 1986). Female college students were observed to score higher on the Eating Disorder Inventory (EDI) (Garner and Garfinkel 1997), and were significantly more likely than those scoring lower to engage in avoidance coping strategies and were less likely to use cognitive behavioural strategies. Pearlin and Schooler (1978) regard mastery as a psychological resource in vitiating stress, in that it gives the individual a sense that they are in control of the forces impinging on them. Skaff et al (1996), view mastery as a global construct, an overarching sense of the general degree to which one experiences control over what goes on in his or her life. Much as one has a belief in

their ability to achieve an outcome, as a personal resource, mastery is viewed as a moderator in the relationship between stress and well being. Whether mastery is a stable characteristic or not is debatable with researchers falling on either side (Cohen and Edwards 1989; Gurin and Brim 1984). Stability is supported by people's ability either to alter aspirations when conditions change or to substitute new roles for old ones, thus sustaining a consistent sense of control. Treating mastery as a stable resource fails to recognise that it may be altered by experience. The conditions under which stability may falter include chronic stress or life transitions that lead to changes in the daily perception of experiences of people's lives. For example, under low stress conditions the challenges of a "normal" day may be perceived as stimulating, whilst under high stress conditions the same challenges may seem unbearable. When such stressors or transitions involve important or salient roles they are especially likely to focus attention on the self and subsequently affect one's self appraisal of mastery. To understand how mastery may be changed as a result of life experiences, we examined it within the context of the stress process described above.

Many factors mediate between a stressor and stress, (Coyne and Downey, 1991: Lazarus and Folkman, 1984). Lazarus indicates that stress is relational and is appraised as taxing or exceeding that individual's resources and as endangering his or her well-being. He refers to coping as the changing thoughts and behaviours which people engage to manage, tolerate or reduce internal or external demands. These coping strategies may or may not be successful in reducing the experience of stress to the individual. Mastery refers to the ability of these thoughts and behaviours to minimise the effects of the stressor (see appendix 1 and 2). An alternative to measuring individual coping strategies is to make a global assessment of whether an individual is

helpless or masterful in negotiating problematic situations. Troop and Treasure (1997) measured helplessness-mastery using semi structured interviews where ratings were based on what was done in comparison to what could have been done. These ratings are based on actual behaviour and take context into account. These authors found that women, who went on to develop an eating disorder, were more helpless and less masterful than were a comparison group of non eating disorder individuals. Furthermore, these differences were also evident in childhood, prior to the onset of an eating disorder (Troop and Treasure 1997b). Psychological sequelae of early life experiences might also contribute to heightened stress responsivity in anorexia nervosa (AN). Dismissive attachments, helplessness and lack of mastery prior to onset of the disorder and avoidant coping response to the triggering event (Troop & Treasure 1997a;Troop & Treasure 1997b) may impair the capacity to manage stressful life events or difficulties in those vulnerable to AN. As a result, these individuals may perceive a uniform stressor as more stressful than their non-anorexic peers, with consequent enhancement of the HPA axis response to the stressor.

Eating behaviour reflects interactions between an individual's physiological and psychological state and environmental conditions (Halmi, 1996). Therefore, this study is exploring the role if any, of mastery and eating behaviour in response to life events. For example, what is the impact of low mastery in restrained eaters? Extensive studies implicate stress and its attendant cortisol secretion in the control of feeding behaviour. Cortisol is known to regulate the HPA axis and stimulate feeding (Epel et al 2001). In conjunction with other factors cortisol enhances a feed forward biochemical system which prevents weight loss. An anomaly arises from the observation that some individuals under stressful conditions lose or maintain weight whilst others gain

weight. There is little information regarding this psychoneuroendocrine response to stress, in the non clinical population. Accordingly, this study has evaluated eating behaviour in a non clinical group.

1.7) Dietary restraint and its development and inhibition.

In the 1970's a new theory of eating behaviour evolved and the term "Restraint Theory" was coined. Restraint theory is based on behavioural processes involved in dieting or restricting food intake. Restrained eating refers to the tendency to cognitively restrict food intake in order to maintain body weight or promote weight loss (Herman and Polivy, 1980). Restrained eaters are individuals who constantly struggle to maintain control over their food intake and weight (Heatherton et al 1988; Lowe, 1993; Shapiro, and Anderson, 2005). The dietary restraint model depicts that a reliance on cognitive control over eating, rather than physiological cues, leaves dieters vulnerable to uncontrolled eating when these cognitive processes are disrupted. Often, eating patterns of restrained eaters are marked by periods of chronic dieting interrupted by episodic bingeing (Polivy and Herman, 1985). Supposedly, any disruption to a restrained eater's self control induces stressful cognitions such as " I've blown it-I might as well continue to eat". This in turn causes eating behaviour to become disinhibited and results in overeating (Ruderman, 1983). Restrained eaters have been reported to disinhibit their eating under stressful situations (Shapiro, and Anderson, 2005).

Previous research has identified disinhibiting agents of restrained eating such as the ingestion of preloads of low and high fat foods (Oliver et al, 2000), alcohol consumption and emotional states encompassing negative affect such as anxiety, depression and worry (Striegel-Moore et al 1998; Telch and Stice, 1998; Wardle et al 2001). However, some evidence exists for restrained eaters who are generally successful in their dietary attempts not to have developed disturbed eating patterns by disinhibition (Westenhoefer, 1991). Therefore, restrained eaters can be subdivided into disinhibited and non-disinhibited restrainers, following an emotionally negative affect laden experience, such as an academic examination (Ogden, 1993).

A frequent finding in ED research into the effects of stress induced eating behaviour is that restrained eaters demonstrate counterregulatory eating behaviours when anxious or stressed (Baucom and Aiken, 1981; Polivy and Herman, 1999; Tanofsky-Kraff et al, 2000; Shapiro, and Anderson, 2004). Distress suppresses eating in unrestrained eaters, and increases eating in restrained eater (Baucom and Aiken 1981, Heatherton, Herman, and Polivy 1991, Heatherton, Polivy, Herman and Baumeister 1993, Polivy and Herman 1999). In many of these studies, the laboratory "taste-test" paradigm has been utilised. Findings using this methodology have identified that restrained eaters paradoxically consume more calories than non-restrained eaters when exposed to emotional stress. Other studies have demonstrated non-restrained individuals consume less food when under stress (Stone and Brownell, 1994). Obese individuals, most of whom tend to be dieters (Herman and Polivy, 1988b), tend to overeat when compared with normal weight individuals, when distressed (McKenna, 1972).

Physical stressors (pain, physical discomfort), are more likely to induce a significant decrease in consumption in unrestrained eaters. Ego threats (e.g. threat to self-image, failure or public humiliation), significantly increase eating in restrained eaters, but have a much less significant effect on unrestrained eaters (Heatherton et al 1991) (see table 1.1. below).

Table: 1.1. The effects of threat type on consumption in restrained and unrestrained eaters.

Threat	**Unrestrained**	**Restrained**
Physical	Consumption reduced (s)	Consumption increased (ns)
Ego	Consumption reduced (ns)	Consumption increased (s)

s = significant
ns = non significant

Heatherton et al (1991)

Many of these studies use laboratory settings in which a Stroop test is seen as a reliable method of inducing an ego-threatening stress (threat to self esteem and emotional stability). Lattimore (2001) examined whether an ego threatening Stroop test could trigger overeating effects similar to those observed using other methods of inducing ego threatening stress (e.g. Polivy and Herman 1999). The ego threatening

Stroop task produced significantly more anxiety and greater ice cream consumption than the film task. Binge eaters consumed significantly more ice cream, and where significantly more anxious following the ego threat task compared to a film task ($p<0.05$). Whilst these laboratory based studies indicate that restrained eaters with low mastery tend to become unrestrained when subjected to an ego threat, The present study determined whether the same finding may be found in the natural environment.

1.7a) Problems with restraint theory.

Restraint theory revolves around the relationship between restriction of food intake and overeating. Accordingly, dieters, bulimics, and bingeing anorexics report episodes of overeating. However restricting anorectics cannot be accounted for within the central tenet of restraint theory, in that they maintain restriction at times when restraint theory would suggest restriction should be reduced or overridden. The discussion in (1.7), supports the observation that dietary restriction results in overeating. Restrained eaters have also been reported to disinhibit their eating under stressful situations (Shapiro, and Anderson, 2005). There are successful dieters who have been obese, dieted to lose weight, and kept this weight off (Klem et al 1998).

What then, are the underlying mechanisms supporting starvation in anorexia? A biological mechanism is proposed by Connan et al (2005), in which argenine vasopressin interferes with negative feedback regulation of CRH at the level of the pituitary gland (p-30). Vegetarians do not eat meat; they also do not binge on meat in response to this exclusion of meat from the diet (Ogden 2003). Reformed alcoholics,

smokers and illicit drug takers no longer consume these substances. These differences may be accounted for through different biological and psychological mechanisms being activated in response to these behaviours. For example, neuroendocrine responses to these changes in food types differ significantly, such as in insulin and cortisol secretion, in activation of the hypothalamic pituitary adrenal response to stress and changes in dopamine secretion in the frontal cortex of the brain.

Food types may need to be separated into low fat/ high fat, low carbohydrate/ high carbohydrate, as the evidence suggests that bingeing in response to stress in dieters results in the consumption of high fat and carbohydrate foods (Wardle et al 2000).

Restraint measures are aimed at only one side of the complex behavioural response seen in conjunction with dietary restraint, e.g. bingeing behaviour, and concern about weight and shape. What are missing are the physiological measures associated with changes in eating behaviour in response to stress. The problem is what physiological measures should be examined. This study suggests that cortisol and its involvement in the stress response would be a logical starting point. Cortisol with its central role in the stress response may link the behavioural response, such as disinhibition of dietary restraint, bingeing, anxiety, depression and other global mental health changes, with physiological responses to stress such as those seen in the neuroendocrine system.

1.8) Mood, anxiety, depression, and body weight

In the previous section, the relationship between dietary restraint, and stress was explored. This section, will focus on the relationship between mood and eating behaviour. Stress affects eating behaviour in some people, but what is the relationship between mood and eating behaviour. How will individuals cope with stress and will their ability to master stress and ways of dealing with it affect their eating behaviour? The discussion will provide an overview of the historical development of theories related to mood and eating behaviour, followed by contemporary literature and research.

1.8.a) A historical perspective

It has been proposed that obese individuals are unable to distinguish between hunger and anxiety, either because they learned to associate them at a young age (Kaplan & Kaplan, 1957), or because they never learned to differentiate between them (Bruch, 1961). It follows that some individuals may perceive the anxiety of a stressor, as if it is hunger and eat accordingly. From these ideas concerning learning history and reinforcement, the psychosomatic theory of stress induced eating evolved. Obese individuals when stressed will be more likely to increase their weight. Conversely, Schachter et al 1968, approached this area of eating behaviour from a different perspective. They suggested that normal weight people had learned to appropriately identify gastric contractions with hunger, whilst overweight individuals have not. Accordingly, normal weight people experiencing stress would reduce food consumption, whilst overweight people would continue to eat. The physiological basis for this internal cue for hunger, is that gastric contractions would reduce in strength

and rate when an individual is stressed (Cannon, 1915; Carlson, 1916), through learned behaviour the normal weight individual would reduce food intake.

Based on the General Effect Model it was proposed that normal weight individuals are sensitive to internal cues such as hunger and gastric motility, whilst overweight individuals are more likely to respond to external stimuli as a cue to eat. Schachter (1971) saw this as a general trait in his obese subjects but did not acknowledge that the obese ate more when anxious. Conversely (Slochower, 1976, 1983; Slochower & Kaplan 1980) replicated Schachters obesity study in an attempt to reconcile Schacters external sensitivity model with psychosomatic theory and concluded that distress results in overweight individuals becoming more sensitive to environmental cues. Therefore, prominent food cues gain control over eating which may easily become excessive.

Heatherton and Baumeister (1991), examined the proposal that ego threats are particularly potent in triggering external sensitivity. They suggested that obese individuals respond to an ego threat by relatively mindless overeating. Dieters faced with a threat to self esteem, may seek to escape aversive self awareness by focussing on salient external stimuli. If that be palatable food, then so be it.

The risk of obesity may increase during times of psychological distress through episodes of disordered eating behaviour such as bingeing. The nature of this relationship continues to be debated by clinicians and researchers and raises considerable controversy. Non clinical studies of obese individuals consistently fail to support the idea that obese individuals differ from their non-obese counterparts in

psychological symptoms, psychopathology, or personality (Stunkard and Sobal, 1995). Nonetheless, studies support the notion that subgroups within the obese population, for example, those presenting for clinical weight-loss treatment and obese binge eaters, show elevated psychopathology (Fitzgibbon et al, 1993; Telch and Agras 1994). Obese eaters who seek treatment show elevated levels of depression (Goldsmith et al, 1992), binge eating, and general psychiatric symptoms (Fitzgibbon et al, 1993).

A survey using questionnaires distributed in libraries, analysed a stratified sample of 90 from 364 returned questionnaires and categorised respondents into under, normal and overweight groups (Geliebter and Aversa 2003). Average age was thirty and equal numbers of male (n = 15), and female (n = 15), were included, in the three groups. This study found that when experiencing negative emotional states or situations, overweight individuals reported eating more than normal weight individuals, and the underweight individuals ate less. For positive emotional states or situations, the opposite occurred, with the underweight group reporting eating more than the other groups.

In a field study, students three to four days before an academic examination showed higher ratings of tension, fear and emotional stress as well as lower ratings of happiness, relaxation and positive mood, on a self rating scale (Macht, et al 2005). The group (n = 40), had a mean BMI 22 (s.d. 2.9). These students also reported a higher tendency to eat in order to distract from stress. It is suggested that eating may distract from the experience of negative emotions (Spitzer and Rodin 1983; Macht, et al 2005).

The results of these studies, coupled with the mounting evidence, that supports the contention that dieting is related to binge eating and that negative mood precipitates disinhibited eating among restrained eaters, but not among nonrestrained eaters requires further investigation. These studies indicate that when attempting to understand the relationship between stress, body weight and symptoms of psychological distress it would be appropriate to explore potential mediating variables, such as dietary restraint, binge eating and food choice. These relationships are explored in this study and presented in fig1.2 (page 21).

1.9) What this study will add to the literature.

This study will combine restraint theory, activational theory and transactional theory in an attempt to unravel the person environment relationship (Lazarus 1991), and its significance to changes in body weight.

These should not be seen as separate mechanisms, rather they form an integrated response to stress. They may even form a spiral effect as a response to a stressor whilst the stressor remains in place. That is to say, disinhibited dietary restraint, increased bingeing and a reduction in mental health may be fuelled by increasing levels of cortisol whilst the stressor remains in place. Therefore rather than a linear model of stress, the psychological and physiological changes which impact on body weight may be associated with a spiral/ circular relationship between these variables as a response to stress (see Appendix 9). A reduction in mental health may result in a further increase in cortisol, further reducing scores of mental health, mastery and coping,

fuelling increases in cortisol secretion and activity within the spiral. This feed forward mechanism, impacting on body weight. The spiral being a transactional response in psychological and physiological antecedents, which are unique to the individual. In (1.6), a schematic view of transactional theory and activation theory is presented. The discussion above outlines the changing person-environment relationships that may impact on body weight. In this study the examination is a stressor present in the environment. In terms of activation theory (Levine & Ursin 1991), the examination as a stressor is the "input", which will challenge the individual. Cortisol secretion as measured in this study in saliva, is synonymous with the significance of the stressor to the individual. For example the more stressful the examination is perceived to be the higher the individual's cortisol secretion. The processing systems present in an individual will filter the impact of the stressor according to transactional theory (Lazarus and Folkman, 1984). For example it is anticipated that individuals with low mastery and high anxiety and depression scores will not filter the impact of the stressor as successfully as those with high mastery and low anxiety and depression scores. The result being, high cortisol secretion (Output: stress response), in the former and lower cortisol secretion in the latter. Therefore the output in terms of the stress response is dependent on the filtering resources of the individual (i.e. level of mastery, anxiety and depression scores).

1.9a) In summary

This literature review has explored the central tenets of eating behaviour that impact on body weight. Clearly, there are many individual attributes, which under certain conditions such as stress will affect body weight. The purpose of this study is to focus on individual attributes of stress reactivity, dietary restraint, binge eating, mood,

mastery and food choice. These attributes will be measured at T_1 baseline (a non stress period), and T_2 a stress period. The information gained should help to elucidate the complex interaction between stress and body weight change. The data should also be relevant to the care and treatment of people with eating problems. With the current increase in obesity there are clear benefits to health from such stress studies, particularly related to cardiovascular and mental health issues.

1.9.b)Features of this study;

- Has a naturalistic and longitudinal design over the course of a stressor and all participants experience the same stressor.
- Measures realistic outcomes in that weight changes rather than simply differences in the amount and type of food eaten are measured.
- Measures individual differences of restraint, cortisol secretion, mastery, anxiety and depression.
- Links psychological and neuroendocrine changes to effects on eating behaviour and weight.
- Measures food choices in response to examination stress in a subgroup of the subjects.

Hypotheses

At Baseline:

1. Body Mass Index (BMI), will be correlated with dietary restraint and bingeing behaviour scores.

In response to the stressor:

2. Salivary cortisol secretion will be elevated and the increase will be associated with a change in Body Mass Index (ΔBMI).
3. Subjects who are high in dietary restraint will become disinhibited and will show an increase in body weight, whilst those low in dietary restraint, will increase in dietary restraint, and show a decrease in body weight.
4. Subjects who are low in mastery will increase their bingeing behaviour.
5. Subjects who increase their bingeing behaviour will show increased consumption of foods that are high in carbohydrate and saturated fats.

Chapter 2.

Methods

This chapter is divided into three main sections, the first deals with the recruitment of subjects and the design of the study. The second section outlines the theory of the biochemical analyses and analytical method and the final section discusses the analysis of the questionnaires and the statistical methods applied to the results.

2.1) Subjects

Information on the study and what would be expected of participants was given to 225 students. A total of 84 individuals agreed to take part in the study, and 71 individuals actually completed the study. They were recruited from information sheets and adverts given to students at The Royal College of Nursing Institute London and latterly at London South Bank University. The criteria for inclusion are that they were female and were undertaking a unit of study, which was assessed by an unseen written examination.

Each individual acted as her own control within the pre and post test design. All participants recruited where undertaking a BSc (hons) Primary Health Care. They held positions as Practice Nurses and were following the degree programme to qualify as Nurse Practitioners. They are mostly self funded or supported by their GP practice partners. Therefore, successful completion of the degree programme was a significant

goal for the participants, and therefore a significant stressor. All participants were undertaking 15 week modules of study culminating in an unseen examination.

Recruitment occurred over three university semesters in order to increase the sample size. The protocol is described in detail in the following sections.

2.2) Study design

The study was designed to assess the hypotheses proposed in Chapter 1.

Time 1.

Participants were recruited in the first week of their module and, in the third week of their module (on a day convenient for them), they were asked to complete the questionnaires and also collect saliva samples, every two hours between 08:00 and 20:00 hrs. The collection of data at this point is referred to as Time 1 (T_1).

Time 2.

In the week of the exam i.e. twelve weeks following Time 1 (T_1), the questionnaires were completed and saliva samples collected. The collection of data at this point is referred to as Time 2 (T_2).

Time 3. (Cohort 1 only)

Individuals were asked to repeat the questionnaires and saliva collection during the summer recess, at a time when they were not taking any exams. The collection of data at this point is referred to as Time 3 (T_3).

The individuals were experienced Nurse Practitioners and measured their own height and weight using their GP practice scales.

2.2.a) Process and time scale

Three cohorts were recruited.	Started	Dropped out
1st cohort February 2001 to May 2001	n = 42	9
2nd cohort February 2002 to May 2002	n = 28	3
3rd cohort October 2002 to January 2003	n = 14	1
Total number of participants was 84	**n = 84**	**n = 13 dropped out**

Number of participants who completed the study = 71

The subjects were asked to complete the following questionnaires;

The Mastery Scale (Pearlin, 1981); The List of Threatening Experiences LTE-Q (Brugha et al 1985), which assesses the individuals exposure to recent life event categories; the General Health Questionnaire-12 (GHQ-12) : (Goldberg, 1972), which assesses global mental health; the Hospital Anxiety and Depression Scale (HADS) : (Zigmond and Snaith, 1983), the Social Questionnaire (Corney and Clare 1985), which

assesses items covering, housing, occupation, finance, social and leisure activities, child-parent and marital relationships, relationships with relatives, friends, neighbours and workmates and legal problems; the Eating Disorder Examination Questionnaire (EDE-Q4) (Fairburn and Beglin 1994), which assesses dietary restraint, weight concern, shape concern and eating concern; the Food Frequency Questionnaire (FFQ: Cade and Margetts, 1988), and our own Visual Analogue Scale assessing the significance of the module being studied and how important it was to the student that they were successful in the examination. More detailed information on these questionnaires including reliability and validity studies are presented below

2.3) Psychometric analysis

2.3.a) Mastery Scale (Pearlin, 1981)

The Mastery Scale asks 7 questions such as;

how strongly do you agree or disagree the following statement

“I have little control over the things that happen to me”.

Respondents are asked to respond to each statement by selecting one of the following Likert Scale responses:

"strongly disagree"(coded 4), "disagree" (coded 3), "agree" (coded 2) or "strongly agree" (coded 1). Here, the higher the score, the greater the sense of mastery, with the exception of questions five and seven which are reverse coded. A total score (per respondent) from the seven items, which equals or is more than 20, is considered having mastery (see appendix 3)

2.3.a.1) List of Threatening Experiences (LTE-Q) (Brugha et al 1985)

A substantial proportion of measured adversity is accounted for by a relatively small group of life event categories covered in one particular inventory. The List of Threatening Experiences Questionnaire assesses 14 life events categories. Whilst the semi structured interview and panel rating technique is much to be preferred, the LTE-Q has been shown to have high test-retest reliability and good agreement with informant information. Concurrent validity, based on criterion of independently rated adversity derived from a semistructured life events interview, making use of the Life Events and Difficulty Schedule (Brown & Harris, 1978: Social Origins of Depression) method showed both high specificity and sensitivity. The LTE-Q is particularly recommended for use in psychological and social studies in which other intervening variables such as social support, coping and cognitive variables are of interest and resources do not allow for the use of extensive interview measures of stress. (see appendix 4)

2.3.a.2) General Health Questionnaire-12 (GHQ-12) : (Goldberg, 1972)

The General Health Questionnaire-12 (GHQ-12) is a self administered screening measure for the detection of minor psychiatric disorder in community and nonpsychiatric clinical settings. The questionnaire is designed to be maximally sensitive to changes in normal functioning and to differentiate psychiatric cases from non psychiatric cases. The GHQ has been shown to be a reliable and valid instrument (Hardy et al 1999) for the detection of minor psychiatric disorder among health workers in the NHS in England. (see appendix 5)

2.3.a.3 Hospital Anxiety and Depression Scale (HADS) : (Zigmond and Snaith, 1983)

The Hospital Anxiety and Depression Scale (HADS), is a reliable, valid and practical tool for identifying and quantifying the two most common forms of psychological disturbances (Herrmann 1997). It was designed with special attention to some specific issues especially relevant for the setting of somatic medicine. The scale was limited to 14 items, which makes it easy to administer and well accepted. The scale is sensitive to mild forms of psychiatric disorder thus avoiding the "floor effect" which is frequently observed when psychiatric questionnaires are used with medical patients. The scale is able to differentiate groups with different prevalences or intensities of anxiety and depression. It allows longitudinal assessments with repeated testing at intervals of about 1 week or more and insensitive to changes in patients emotional state. It is well documented to predict mood over intervals of 1 year and longer. It also predicts compliance, quality of life (HADS depression), and physical symptoms (HADS anxiety). (see appendix 6)

2.3.a.4) Eating Disorder Examination-Questionnaire, (EDE-Q4) (Fairburn and Beglin 1994)

Compared to an investigator-based interview, the Eating Disorder Examination (EDE), with a self-report questionnaire (EDE-Q4), based directly on that interview for assessing the features of eating disorders. Participants were a community sample of 243 women (aged 16-35 yrs) and a patient sample of 23 women with bulimia nervosa (mean age 24.3 yrs) and anorexia nervosa (mean age 15.8 yrs). The EDE-Q4 has been evaluated and excellent internal consistency and test retest reliability for the 4 subscales , restraint, weight concern, shape concern and eating concern have been

reported (Luce and Crowther, 1999), supporting the psychometric adequacy of the questionnaire. For the purposes of this study only the restraint scale and binge score were used (see appendix 7).

2.3.a.5) Food Frequency Questionnaire (FFQ).

This 63 item questionnaire comprises food items grouped into six categories (dairy products; meat and fish; breads and cereals; fruit and vegetables; beverages; miscellaneous). Participants are required to note how often they eat each item on a six point scale ranging from "two or more times a day" to "rarely/ never". Higher scores relate to less frequent consumption of the item/ category in question. Dietary intake of items such as fibre, food energy, saturated fat, polyunsaturated fat, total fat, protein, sugar, starch and other carbohydrates are then calculated for each individual. The questionnaire has a test-retest reliability of $r = 62$ ($p<.01$: Cade and Margetts; Margetts, Cade and Osmond, 1989) (see appendix 8).

Table 2.1. Psychometric and biometric measurements taken during the study.

	T1 N = 84	T2 N = 71*	T3 N = 18*
Salivary Cortisol (AUC)	✓	✓	✓
Restraint (EDE-Q)	✓	✓	✓
BMI	✓	✓	✓
Binge (EDE-Q)	✓	✓	✓
GHQ-12	✓	✓	✓
HAD-anxiety	✓	✓	✓
HAD-depression	✓	✓	✓
HAD-total	✓	✓	✓
List of Threatening Experiences	✓	✓	✓
Mastery	✓	✓	✓
EDE-Q Total	✓	✓	✓

*13 failed to complete the study

**Only the first cohort completed this final assessment

Cohort 2 and 3 completed The Food Frequency Questionnaire, as described on page 77 (Cade and Margetts; Margetts, Cade and Osmond, 1989).

Table 2.2. Food Frequency Questionnaire completed by 38 subjects.

	T1 N = 42	T2 N = 38	Dropped Out N = 4
Food Frequency Questionnaire (FFQ)	✓	✓	

This questionnaire was included in cohort 2 and 3 only, to explore changes in food choice during the stress period. The total number of participants who completed this questionnaire was 38. The 38 participants completed all of the questionnaires listed in Table 2.2 above.

2.4) Biometric: BMI

BMI was measured at time 1 and 2.

2.5) Biochemical analysis

2.5.a) Introduction

Saliva cortisol sampling provides a reliable and stress free estimation of circulating free cortisol concentrations (Laudat et al 1988). All participants were instructed to collect saliva samples at 2 hourly intervals from 8am until 8pm during the course of a normal day. Samples were obtained using standard salivettes (Sarstedt, Leicester). Saliva samples were refrigerated after completion until they could be frozen and stored at -70^{0}C at The Institute of Psychiatry. The samples were then transported to The Royal Infirmary in Glasgow packed in dry ice and then stored at -20^{0}C, prior to assay. All samples where analysed in the Biochemistry Dept of The Royal Infirmary Glasgow using an in house RIA technique. (described below).

2.5.a.1) Radioimmunoassay (RIA).

RIA is a long established technique for the measurement of low concentrations of analyte in solution, and utilises an antibody raised against the analyte, a radiolabelled analogue of the analyte and a method of separating the antibody-radiolabelled analogue from unbound radiolabelled analogue at the end of the assay.

2.5.a.2) Antibody.

The antibody is produced by immunising an animal (typically a rabbit) with the analyte which has to be sufficiently large to elicit an antigenic response: if it is a small molecule, such as a steroid, it can be coupled to a hapten, for example albumin, to provide a suitable epitope for antibody production.

The antibody is tested for its binding affinity to similar hormones and the percentage cross reactivity of each compound is determined. The binding of these compounds may vary across a range of concentrations accordingly more than one concentration is tested. The percent cross reactivity is defined as the concentration of analyte causing 50% displacement, multiplied by 100% (Abraham, 1969). Similarly the affinity of the antibody to the different isoforms of the hormone can be determined. To summarise, an ideal antibody for RIA binds to all isoforms of the hormone with equal affinity and does not bind to compounds of a similar structure which may be present in the sample.

2.5.a.3) Tracer.

A radiolabelled analogue of the analyte (i.e. the tracer) is required, in all analyses in this study iodine 125 (^{125}I) labelled analogues were used which gives tracers of high specific activity (i.e. high levels of radioactivity per unit mass of radiolabelled analyte). A variety of methods can be used to attach this isotope to the analyte and care must be taken not to significantly alter the antigenic properties of the molecule. This can be a problem with small molecules, such as steroids as the iodine residue is relatively large in comparison to the steroid structure and can deform it. To minimise this problem, ^{125}I is attached via a bridging molecule such as *N*-hydroxy-succimide:

this bridging molecule should not be identical to the molecule used to attach the analyte to the hapten for antibody production, otherwise the antibody may bind to the tracer with higher affinity than it binds to the analyte.

2.5.a.4) Standardisation.

The assays are standardised using solutions of known concentration and these are prepared by two methods, a) addition of pure analyte free serum, or b) comparison of the concentration of the analyte in the assay standards with a standard reference preparation. The latter method is used for many peptide hormones, as the hormone may exist as a number of isoforms of differing biological potency. The activity of the standards are determined by bioassay (see, Robertson et al, 1987, for a review) and compared with the activity of a reference standard under the same assay conditions.

The standard curve in an assay is typically derived from five or six standards of increasing analyte concentration in addition to the "zero" standard.

2.5.a.5) Separation methods

The antibody – analyte or tracer complex from unbound analyte or tracer can be separated by a variety of methods. A common method is precipitation of the antigen – antibody complex with a second antibody raised against serum from animal species used to raise the first antibody. Typically a rabbit is used to produce the first antibody and a donkey or goat to raise the second. Precipitation of the complex can be assisted by volume expanders, such as polyethylene glycol (PEG), these act by restricting the

volume of solution available to the antibodies, forcing them into closer association and therefore increasing the rate of binding and assisting in precipitation of the antibody complexes. Centrifugation is then used to produce a pellet which can be counted in a gamma counter.

2.5.a.6) The theory of RIA.

RIA is based on the competition of analyte and tracer for antibody binding site in the assay tube. In samples with a low concentration of analyte, relatively more tracer will be bound and with a high concentration of analyte, relatively less tracer will be bound and therefore, the amount of radioactivity in the precipitated pellet is inversely proportional to the concentration of analyte in the sample.

2.5.a.7) RIA assay procedure.

Table 2.3 shows a typical procedure for a RIA (page 87).

Table 2.3. Sample and reagent addition flow sheet for a typical RIA.

	Tracer	**First antibody**	**Second antibody**	**Sample**
Total counts	Yes	No	No	No
NSB	Yes	No	Yes	Zero standard
Zero standard	Yes	Yes	Yes	Solution containing none of the analyte
Standards	Yes	Yes	Yes	Solution with known concentration of analyte
Samples	Yes	Yes	Yes	Subject samples or quality control material

NSB = Tubes assessing non specific binding tubes.

2.5.a.8) Standard curve calculation method.

The 4 parameter logistic method was used, this includes the zero standard and the NSB in the calculation. The curve equation shown below gives a sigmoid curve of bound counts against log concentration.

$$Y = (a - d)/ (1 + (x/c)^b) + d$$

Where y = bound counts; x = concentrations; a = response of zero standard; b = - the slope of the log – logit plot; c = ED50 [the concentration at which half maximal binding of tracer (maximal is the amount bound to the zero standard) occurs]; and d = response for the NSB. The curve is then fitted using the method of least squares fit.

2.5.a.9) Quality control (QC) systems.

The study was conducted at three time periods over two years, this generated a large number of samples ~ 1260. Clearly this number is more than could be practicably assayed at one time. Also the effects of time when stored for a long period are unknown. Therefore the samples from each cohort where analysed as soon as possible after the second samples at time two had been collected.

2.5.a.10) Preparation of samples & cortisol measurement

Saliva specimens were stored frozen (-20˚C) on receipt at the laboratory (Department of Clinical Biochemistry, Mcewen Building, Glasgow Royal Infirmary). Before analysis samples were thawed and mucins were precipitated from the specimens by centrifugation at 2500rpm (at 4˚C) for ten minutes. Cortisol was measured in the clear supernatant fraction by an 'in house' radioimmunoassay using a method developed solely for the use of cortisol measurement in saliva (McConway and Chapman 1986; McCartan, Lamey and Wallace 1996). The radioimmunoassay utilises a microencapsulated antibody and ^{125}I-cortisol as tracer(1). The assay requires 100µl of saliva and samples are measured in duplicate. The assay sensitivity is 0.9mol/L, an inter-assay coefficient of variability less than 10%; and an intra-assay coefficient of variation less than 15%. The assay range is 1-32nmol/L as interpolated from the precision profile (i.e. the points where the coefficient of variation was less than 22%)(2). Salivary cortisol concentrations greater than 32nmol/L were diluted with assay buffer and reanalysed. Any visual blood contamination was noted and the result withheld if a high result (>32nmol/L) did not dilute in a linear fashion – inferring blood contamination. Three internal quality control assessment pools were prepared by pooling specimens of varying concentrations that covered the assay range (e.g. 4, 10 and 15nmol/L). All three pools were run in each assay to assess assay performance.

2.5.a.11) Area under the curve (AUC)

AUC is calculated using the following formula, which is the trapezoid rule.

AUC = 0.5 X 1st time point ($08:00h^{-1}$) + sum of 2nd to penultimate time point + 0.5 X last time point ($20:00h^{-1}$).

2.6) Statistical analysis

2.6.a) Preliminary Analysis

Normative descriptive statistics were performed.

Mean, standard deviation and T-Test data of the main predictors in the study and the dependent variable (BMI), for the 71 individuals who completed the study and the 13 who dropped out after T_1 are shown in Table 3.1.1. page 100

Cronbach alpha scores for the measurement scales used in the study are shown in Table 3.1.2 (see page 104).

Correlation matrix of the main variables measured in the study and the change in these variables at T_2 are shown in Tables 3.1.4 and 3.1.5 (see pages 107 & 112)

2.6.a.1) Main analysis

Linear regression analysis predicting the effects of independent variables on the dependent variable change in Body Mass Index (Δ BMI) were performed to build a theoretical model of the study. Variables and effects explored in the study included

2.6.a.2) Predictor variables

	(abbreviation)
Cortisol/ Change in cortisol secretion	/ ΔAUC
Restraint/ Change in restraint	/ ΔRestraint
FFQ/ Change in food choice e.g. fat, carbohydrate	/ ΔFFQ
Binge/ Change in bingeing behaviour	/ ΔBinge
Mastery/ Change in mastery	/ ΔMastery
Anxiety/ Change in anxiety	/ ΔAnxiety
Depression/ Change in depression	/ Δ Depression

The change referred to is the change in the measurement of the above variables between T_1 and T_2.

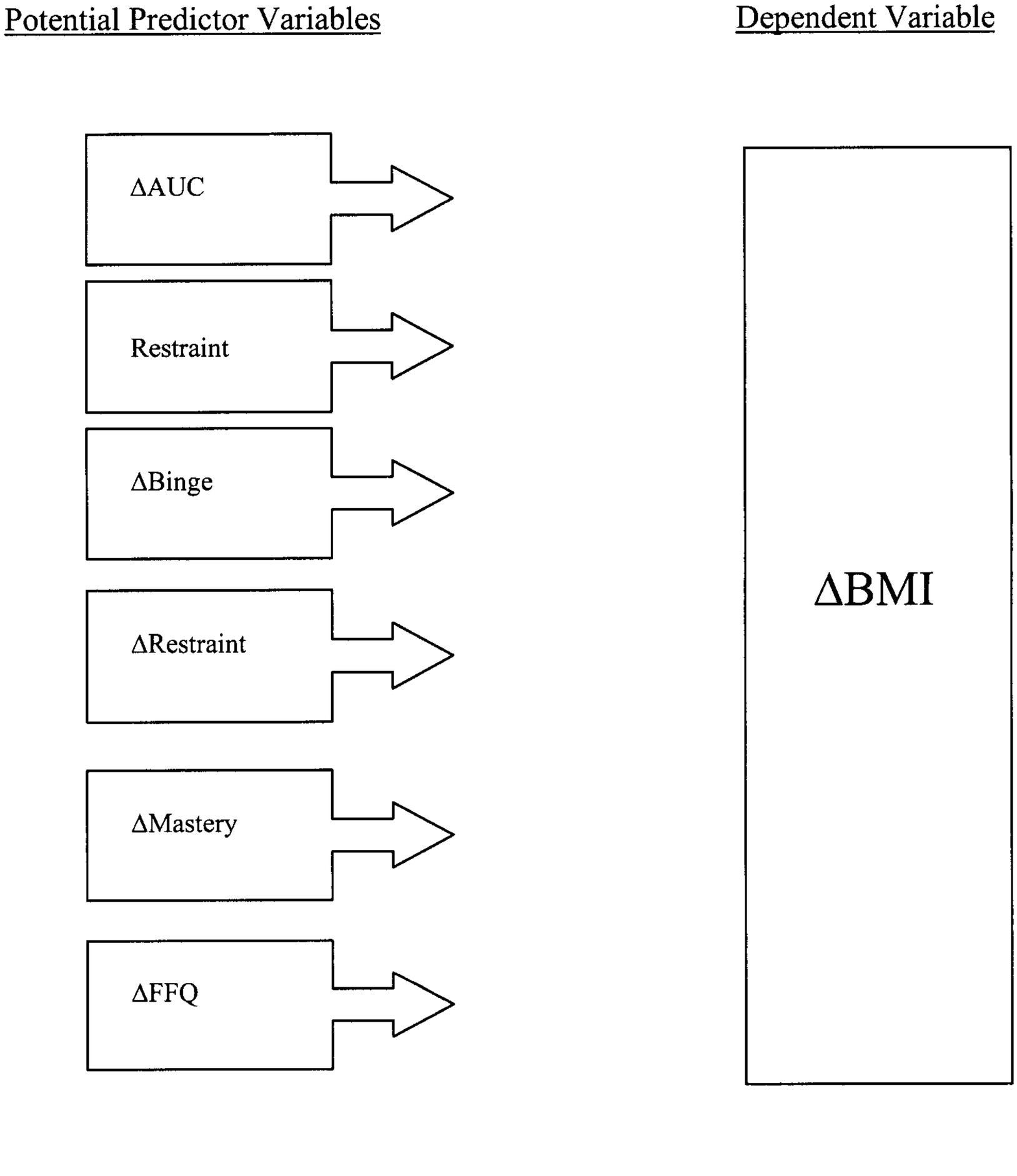
2.6.a.3) Dependent Variable

Change in Body Mass Index $\frac{\text{Weight Kgs}}{\text{Height}^2}$ = Δ BMI

2.7) Model development and linear regression analysis.

The diagram below shows potential variables and the dependent variable in the study.

Fig 2.1 The potential predictor variables, that may have direct main effects on the dependent variable (ΔBMI).



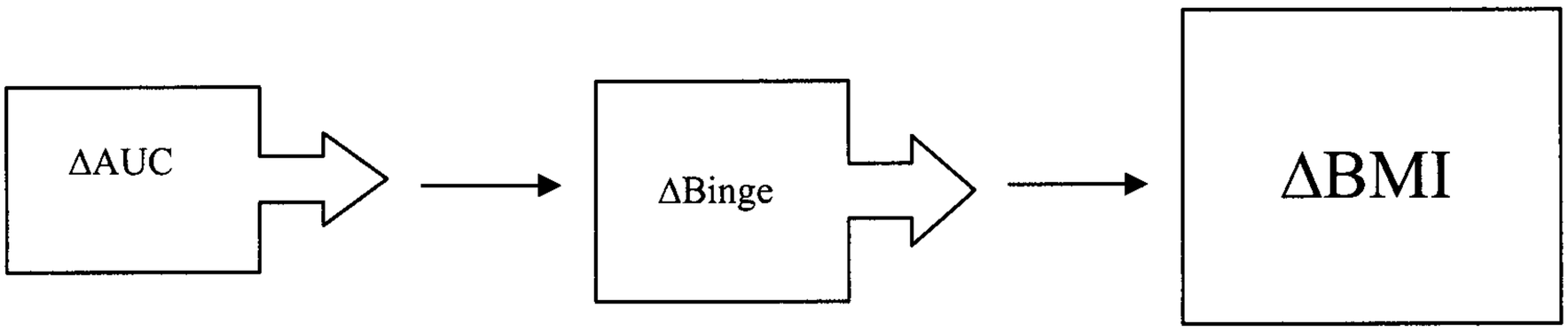
2.7.a) Multiple regression was used to explore mediators and moderators.

2.7.a.1) Potential mediator effects.

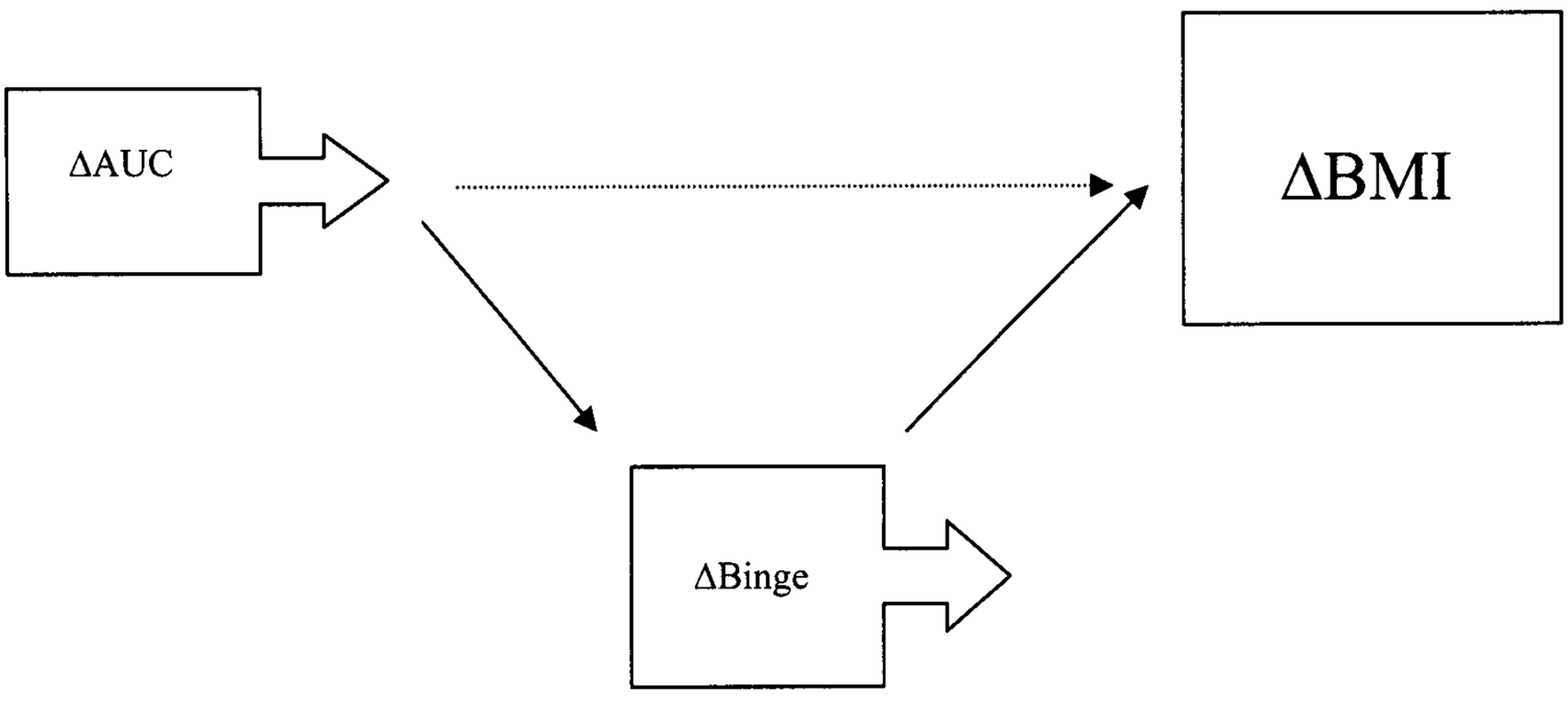
A variable is said to mediate the relationship between a predictor and an outcome variable if the predictor variable first has an effect on the mediator variable and this in turn influences the outcome variable (Baron and Kenny, 1986). The moderator or predictor variable, from statistical evaluation may be shown to influence changes in the dependent variable. However a change in the dependent variable, may not be directly attributable, to the impact of the predictor or moderator variable. For example, any change in body weight (ΔBMI), may not be directly attributable to a change in cortisol secretion during the stress period (ΔAUC). The ΔBMI may arise as a result of a change in eating behaviour such as binge eating (ΔBinge), and binge eating may emerge as a behavioural change in response to a rise in cortisol. In this example binge eating may be a mediator and the next fig (2.2), depicts these variables as a model. This model explores the notion that binge eating is a mediator between the predictor variable ΔAUC and the dependent variable ΔBMI. Any of the predictor variables in the fig 2.1 above could be mediators. The use of multiple regression analysis will enable a model of mediator variables to be constructed. Fig 2.2 below describes the relationship between predictor, mediator and dependent variables.

Fig 2.2 The relationship between the predictor variable ΔAUC, the potential mediator ΔBinge, and the dependent variable ΔBMI

Complete (or perfect) mediator



Partial mediator



Multiple regression analysis will identify a mediator (Baron and Kenny, 1986) if the following 3 steps can be established:

1. ΔAUC is a significant predictor of ΔBMI
2. ΔAUC is a significant predictor of ΔBinge
3. ΔBinge is a significant predictor of ΔBMI when controlling for ΔAUC

If ΔBinge is a complete (or perfect), mediator of the relationship between ΔAUC and ΔBMI, the effect of ΔAUC, when controlling for ΔBinge, should be zero.

If ΔBinge is a partial mediator of the relationship between ΔAUC and ΔBMI, the effect of ΔAUC, when controlling for ΔBinge, should be reduced but not eliminated.

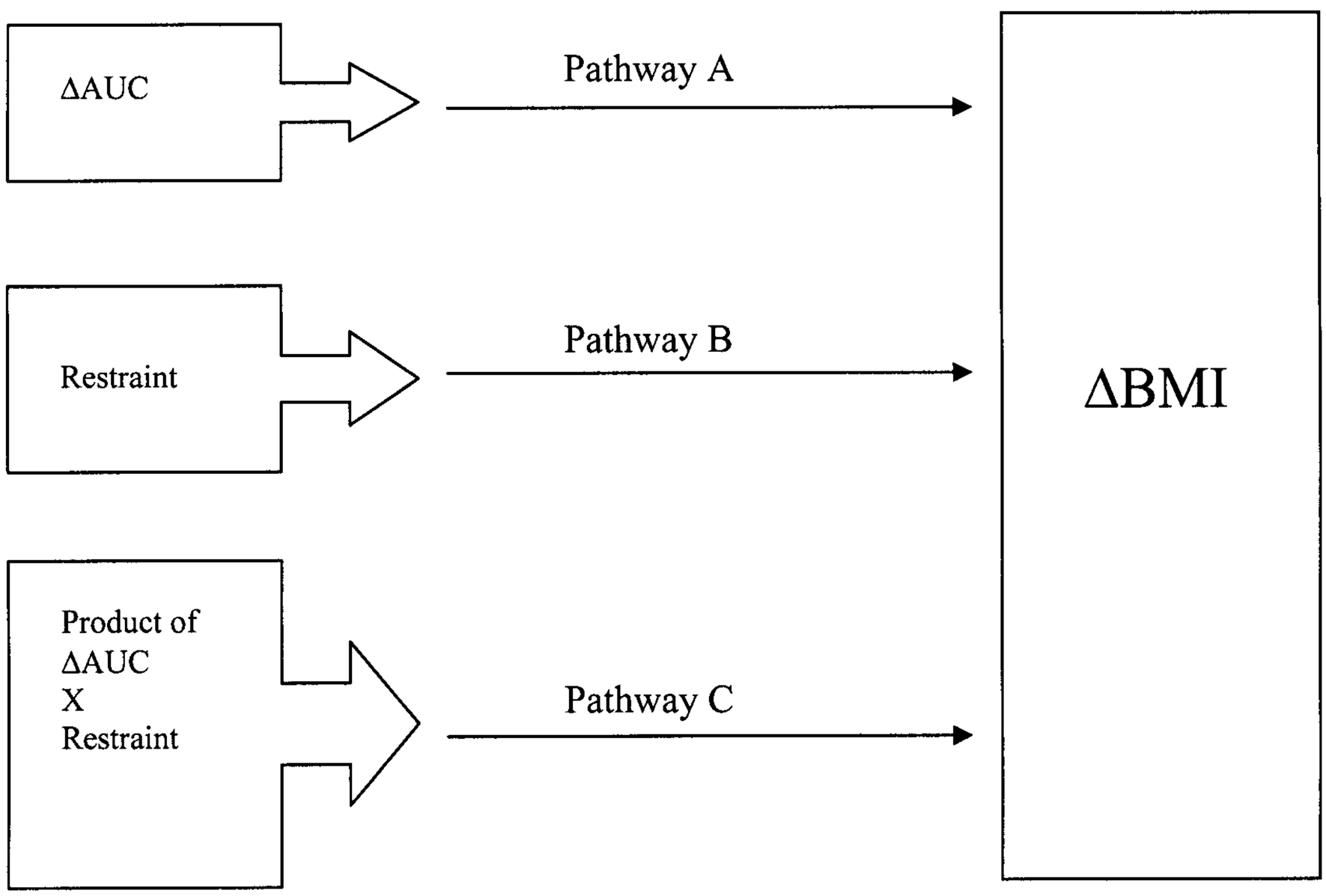
(To determine by how much the effect of ΔAUC has been reduced, subtract the standardized β-weight of ΔAUC when controlling for ΔBinge from the standardized β-weight of ΔAUC when not controlling for ΔBinge).

2.7.a.2) Potential moderator effects.

Baron and Kenny (1986), describe a moderator is a variable that affects the direction and/ or strength of the relation between an independent or predictor variable and a dependent variable. Fig 2.3 below depicts three causal paths that feed into the outcome variable of ΔBMI. The impact of change in cortisol secretion (ΔAUC), as a predictor (path a), the impact of restraint as a moderator (path b), and the interaction or product of these two product of ΔAUC X Restraint (path c). The moderator hypothesis is supported if the interaction (path c), is significant. Significance indicates that with the moderator in the model the strength of change in the dependent variable is weakened. There may also be significant main effects for the predictor and the moderator (paths a and b), though these are not directly relevant conceptually to testing the moderator

hypothesis. However these main effects can be impressive at a statistical and conceptual level and may have considerable impact on the criterion variable. In this study, ΔAUC and restraint may both have an impact on ΔBMI.

Fig 2.3 The diagram shows that the predictor variables ΔAUC and restraint have direct main effects on the dependent variable ΔBMI, and Product of ΔAUC X Restraint as a potential moderator.



2.8) Summary of statistical analysis

A series of multiple regression analyses will explore the predictive effects of the above variables on ΔBMI. Mediator and moderator effects of the potential predictive variables on the dependent variable ΔBMI are presented in a series of tables. (see Chapter 3). A theoretical model is built from this information (see Fig 1.2 page 21).

The following series of regression analysis will be presented in the results section.

In the whole group n = 71

Predicting ΔBMI: Main and moderating effects of stress and restraint.

Predicting ΔBinge: Main and moderating effects of stress and restraint.

Predicting ΔBMI: Main and moderating effects of stress and restraint, with potential mediator ΔBinge.

Predicting ΔRestraint: Main and moderating effects of stress, restraint T_1 and ΔBinge.

Predicting ΔBMI: Main and moderating effects of stress, restraint T_1, ΔBinge and ΔRestraint.

In a subgroup n = 38. (in this group Food Choice was measured at baseline and during the stress period.

Predicting ΔBMI: Main and moderating effects of stress and restraint.

Predicting ΔFFQ: Main and moderating effects of stress and restraint.

Predicting ΔBMI: Main and moderating effects of stress, restraint and food choice.

Chapter 3

Results

Introduction to Chapter 3 results

In this chapter the data will be presented in three parts

3. 1. This section present the mean, standard deviations, significance of T-tests and bivariate correlations of all variables measured in the 71 subjects who completed the study. This is presented along with data of the 13 subjects who took part at time 1 but failed to complete the study.

3.2. This section presents linear regression analysis of predictive variables identified from the bivariate analysis. Predictive variables are then explored in terms of their effects on the dependent variable change in BMI (ΔBMI). Potential mediators are then evaluated for mediational effects on the dependent variable ΔBMI. This information is used to generate a model of the effects of predictor variables, mediators and moderators on the dependent variable (ΔBMI).

3.3. A sub group of n = 38, individuals from the main study (n = 71), completed a Food Factor Questionnaire. Information is presented regarding food choices and total kilocalories consumed at both time periods.

3) Introduction

A total of eighty four individuals took part in the study with variables being measured at commencement of the study time 1 (T_1), and during the week of their exam before the exam day, time 2 (T_2). The study was conducted in three phases, i.e. there were three different cohorts (see study design page 75). In the first cohort, there was a third time point (T_3). which occurred at least six weeks after (T_2) 18 subjects in cohort 1 completed $T_{1, 2}$ & $_3$. Seventy one individuals from the three cohorts, completed the study by returning questionnaires and saliva samples at T_1 and T_2. Thirteen individuals did not return either questionnaires or saliva samples or both.

Section 3.1

3.1) Normative data

This section will present descriptive statistics and show variables, which are correlated with each other. Correlations will inform the regression analyses which will be presented in section 3.2.

3.1.a) Comparison between measurements at time 1(T_1) and time 2 (T_2).

Table 3.1.1: Mean and standard deviation of the main predictors in the study and the dependent variable (BMI), for the 71 individuals who completed the study and the 13 who dropped out after time 1.

	T_1 Mean (SD) n = 71	T_2 Mean (SD) n = 71	t	Significance (2 tailed) T_1 vs T_2	Did not complete T_2 n = 13
Height	1.63 (0.07)				1.65 (0.07)
Age	42.95 (7.1)				44.16 (7.8)
AUC	77.33 (26.55)	92.83 (30.98)	-6.20	0.001	58.88 (20.47)
Restraint	1.77 (1.29)	1.21 (1.01)	6.22	0.001	1.85 (1.34)
BMI	25.23 (4.26)	25.55 (4.48)	-4.22	0.001	25.72 (3.62)
Binge	0.49 (0.73)	1.32 (1.84)	-4.32	0.001	0.54 (0.66)
GHQ	2.61 (3.00)	4.27 (3.51)	-4.54	0.001	2.15 (3.46)
HAD anx	7.5 (3.9)	8.9 (4.5)	-4.31	0.001	7.8 (4.1)
HAD dep	3.9 (3.3)	5.3 (3.8)	-4.68	0.001	4.5 (3.9)
HAD Total	11.4 (6.6)	14.3 (7.8))	-5.07	0.001	12.3 (6.94)
LTE	0.86 (1.10)	0.95 (1.35)	-0.87	0.40	1.08 (1.75)
Mastery	21.25 (3.14)	20.51 (3.32)	2.61	0.01	21.08 (1.61)
EDE-Q4 Total	2.31 (1.2)	2.0 (1.15)	2.02	<0.05	2.6 (1.25)

Table 1 shows the means and standard deviations of the main variables in the study at T_1 and T_2 and the significance of a two-tailed T-test. For comparison, the means and standard deviation of the 13 who only completed T_1 is also shown.

3.1.a.1) Discussion of means and t-test data

Salivary cortisol as measured by Area Under the Curve (AUC) nmol/l was 77.33 nmol/l (26.55) at T_1 and increased to 92.83 (30.98) at T_2. This increase was significant at $p < .001$. An increase in cortisol secretion of this magnitude and significance confirms that the subjects were experiencing a more stressful environment at T_2, than T_1. The design of the study would support the examination as being a significant stressor.

The global EDE-Q4 score decreased by 11% at T_2 to 2.0 (1.15), from 2.31 (1.2), and this was significant at $p < .05$. Previously, a large community sample of healthy women (n = 208), scored a mean of 1.55 (s.d.1.21) (Fairburn and Beglin 1994; Chris Fairburn, Frances Connan, personal communication)). In a study by Frances Connan (personal communication), a healthy group of women scored a mean 1.3 (0.5 – 3.9), and another study has reported a mean of 1.42 (s.d. 1.04 Mond et al 2004), n = 195 age 18 – 45. Therefore, the measurement of global eating pathology in this study falls within the limits expected in the study sample.

In a study of patients with binge eating disorder (BED), n = 82, mean age of 42 (S.D 9.8) the total EDE-Q4 score was 23.5 (Grilo, Masheb and Wilson 2001). Fairburn (1994), has found scores in the range of 0.25 – 17.8 in recovered anorectics and 3.5 – 33 in women suffering with anorexia nervosa. Further supporting the reliability in measurement of global eating pathology in this study.

Dietary restraint, as measured by the EDE-Q4, is 32% lower at T_2 to 1.21 (1.01), from 1.77 (1.29), and this is a significant decrease $p < .001$. Restraint as measured by the EDE-Q4 in our sample was found to be 1.77 (s.d. 1.29) n = 71. Previously a large community sample of healthy women (n = 208), scored a mean of 1.25 (s.d.1.31) (Fairburn and Beglin 1994; Chris Fairburn, Frances Connan, personal communication)). Another study reported a mean of 1.29 (s.d. 1.27 Mond et al 2004), n = 195 age 18 – 45. In a study of patients with binge eating disorder (BED), n = 82, mean age of 42 (S.D 9.8) the dietary restraint score was 13.7 (Grilo, Masheb and Wilson 2001). The measurement of dietary restraint in this study therefore falls within the limits expected in the study sample.

Binge as measured by the EDE-Q4 was significantly higher at T_2 than at T_1. The increase of 170% was significant at $p < .001$. Binge as measured by the EDE-Q4 in our study had a mean of 0.49 (s.d. 0.73) at baseline and 1.32 (1.84) during the stress period. In a study of patients with binge eating disorder (BED), n = 82, mean age of 42 (S.D 9.8) the binge score was 2.5 (Grilo, Masheb and Wilson 2001). The present study scores are below that of a group of similar aged women with binge eating disorder.

The mean BMI measured at T_1 was 25.23 (s.d. 4.26). There is an increase in Body Mass Index (Δ BMI), at T_2 to 25.55 (s.d. 4.48), of 1.3% and this is significant at $p <$ .001. The range of normal body weight is currently accepted internationally as a BMI of 18.5-24.9 kg/m^2 .The World Health Organisation classifies a BMI ≥ 25 kg/m^2 as overweight, for an adult and a BMI ≥ 30 kg/m^2 as obese. Andres (1995), provides a range for BMI which increases with age, given the mean age of our sample (42 yrs), the mean BMI is within the expected range.

Global mental health pathology as measured by the GHQ-12 scale increased from 2.61 (s.d. 3.00) at T_1 to 4.27 (s.d. 3.51) at T_2 and this was significant at $p < .001$. Respondents with a score of 4 or more are classified as possessing "caseness". This threshold cut off point, and the 12-item scale of the GHQ-12 has been employed in a number of other studies (Pajak et al 2003; Calnan et al 2002; Walsh and Walsh 2002). These and other studies used the cut off point of 4 concluding that subjects with a score of 4 or more may be suffering psychological ill health. The present study confirms that during the stress period, with a mean score of 4.27, many subjects may be suffering from psychological ill health. This finding has implications for the use of unseen written examinations as a form of academic assessment.

Anxiety as measured by the HAD scale increased from 7.5 (s.d. 3.9) at T_1 to 8.9 (s.d. 4.5) at T_2 and this was significant at $p < .001$. Depression as measured by the HAD scale increased from 3.9 (s.d. 3.3) at T_1 to 5.3 (s.d. 3.8) at T_2 and this was significant at $p < .001$. Snaith and Zigmond (1994), recommend a cut off point of HADS-A and HADS-D scores of 8 or over to classify possible cases of clinically relevant levels of anxiety and depression respectively. The HADS scores therefore show anxiety and depression to be increased at T2 with anxiety levels reaching into clinically relevance and depression increasing but not clinically relevant.

Mastery as measured by the Mastery Scale was reduced by 3.5 % at T_2 and this was significant at $p < .01$. The scale has 7 items, answered on a 4-point (strongly agree/disagree) scale. Scores range from 7 (low mastery) to 28 (high mastery). The present scores of 21.25 and 20.51 indicate that the study individuals were highly

masterful. Pearlin and colleagues (1981) indicate a correlation of .44 between time 1 and time 2 measures, our correlation was .72 $p < .01$. In the current study, this scale had relatively high reliability; Alpha was calculated as .79. Previous studies have found scores of 8 to 35, with an Alpha of .82 (Greenberg and Grunberg, 1994).

3.1.a.2) Cronbach Alpha scores

The table below shows Cronbach Alpha scores for the measurement tools used to measure the main variables in the study. These are a measure of internal consistency

Table 3.1.2: Cronbach Alpha scores for the main variables in the study.

	Cronbach Alpha
Height	single item
AUC	.95
Restraint	.75
BMI	single item
Binge	single item
GHQ	.90
HAD Total	.91
LTE	.60
Mastery	.84
EDE-Q4 Total	.93

All measures used show very good internal consistency. This was to be expected based on reliability and validity studies conducted for each measure. (Single item scales do not have an internal consistency, as there is only one item).

3.1.a.3) Comparison of the 13 subjects who did not complete T_2

Table 3.1.3: T_1 scores of those who did not complete the study n = 13 with the 71 individuals at T_1 that completed the study.

	T_1 Mean (SD) n =7 1	Did not complete T_2 n = 13
Height	1.6 (0.07)	1.65 (0.07)
AUC	77.3 (26.6)	58.9 (20.5)
Restraint	1.8 (1.3)	1.9 (1.3)
BMI	25.2 (4.3)	25.7 (3.6)
Binge	0.49 (0.73)	0.54 (0.66)
GHQ	2.6 (3.0)	2.2 (3.5)
HAD anx	7.5 (3.9)	6.8 (3.4)
HAD dep	3.9 (3.3)	3.2 (3.1)
HAD Total	11.4 (6.6)	10.0 (6.5)
LTE	0.86 (1.10)	1.08 (1.75)
Mastery	21.3 (3.1)	21.1 (1.6)
EDE-Q4 Total	2.3 (1.2)	2.5 (1.5)

The largest differences observed in this group compared with the main study group were, a lower cortisol secretion (24%), and a lower GHQ score (15%), and higher LTE score (25%).

3.1.a.4) Summary

In accord with our expectations and hypotheses, the subjects in the main study were under significantly more stress at T_2. This could be attributed to the examination that they would be sitting within a few days of the sample being taken. At T_2 binge eating increased along with an increase in BMI. Interestingly, there was a reciprocal fall in the mean restraint score and the EDE-Q4 as a global measure of eating behaviour. Both global mental health pathology and anxiety and depression increased at T_2. In summary, the examination was a significant stressor. The data shows that as stress increased (AUC), restraint decreased with a reciprocal rise in binge eating and mental health pathology that resulted in an increase in body weight. A significant reduction in mastery at T_2 was noted with a reciprocal increase in bingeing and depression. These observations will be explored in the next section using bivariate correlations at T_1 and T_2 and bivariate correlations of changes at T_2.

3.1.b) Bivariate analysis.

Bivariate correlations were used to investigate the strength of the relationships among variables in the baseline assessment T_1, and at T_2, (Table 3.1.4).

Table 3.1.4: Bivariate correlations of all study variables at T_1 and T_2.

		BMI	AUC cortisol	Dietary Restraint EDE-Q4	Binge Eating EDE-Q4	EDE-Q4 Global	mastery	GHQ
BMI	T_1							
	T_2							
AUC	T_1	.07, $p = .59$						
cortisol	T_2	.09, $p = .47$						
Dietary	T_1	.34, $p < .01$**	-.11, $p = .38$					
Restraint	T_2	.19, $p = .11$	-.08, $p = .52$					
EDE-Q4								
Binge	T_1	.18, $p = .14$	-.24, $p < .05$*	.28, $p < .05$*				
Eating	T_2	.49, $p < .001$**	.03, $p = .78$	.08, $p = .49$				
EDE-Q4								
EDE-Q4	T_1	.48, $p = .001$**	-.006, $p = .96$	.57, $p < .001$**	.44, $p < 0.001$**			
Global	T_2	.33, $p = .01$**	-.05, $p = .67$	.55, $p < .001$**	.55, $p < 0.001$**			
Mastery	T_1	-.25, $p < .05$*	-.11, $p = .35$	-.20, $p = .10$	-.15, $p = .22$	-.39, $p < .001$**		
	T_2	-.40, $p < .01$**	.04, $p = .75$	-.05, $p = .71$	-.29, $p < .05$*	-.27, $p < .05$*		
GHQ	T_1	.01, $p = .93$	.06, $p = .96$	-.12, $p = .32$	.12, $p = .34$	.23, $p = .06$	-.36, $p < .01$**	.
	T_2	.18, $p = .14$	-.10, $p = .43$	-.05, $p = .66$	.11, $p = .35$	.14, $p = .24$	-.51, $p < .001$**	
HAD	T_1	.15, $p = .21$	-.02, $p = .86$	.09, $p = .43$	.10, $p = .43$	.28, $p < .05$*	-.49, $p < .001$**	.75, $p < .001$**
Total	T_2	.24, $p < .05$*	-.03, $p = .79$	.04, $p = .72$	.24, $p < .05$*	.23, $p < .05$*	-.50, $p < .001$**	.81, $p < .001$**

Table 2 Baseline Bivariate correlations T_1 and T_2 n = 71.
**. Correlation is significant at the 0.05 level (2 tailed).*
***. Correlation is significant at the 0.01 level (2 tailed).*

3.1.b.1) Discussion of bivariate analysis.

3.1.b.2) Correlations at T_1.

High BMI is strongly correlated with high scores in global eating behaviour (EDE-Q4) ($p < 0.001$), including restraint ($p < 0.01$), and low scores in mastery ($p < 0.05$),.

Cortisol secretion increases as bingeing decreases ($p < 0.05$).

High restraint is strongly correlated with global eating behaviour scores ($p < 0.001$), and correlated with binge eating ($p < 0.05$).

High scores in global eating behaviour, bingeing and restraint are observed ($p < 0.001$), coupled with anxiety and depression ($p < 0.05$) as measured by the HAD scale, and low scores of mastery ($p < .001$).

High mastery is observed with low anxiety and depression ($p < 0.001$), as measured by the HAD Scale and with the global score for mental health as measured by the GHQ-12 ($p < 0.01$).

As would be expected, a high global score for mental health as measured by the GHQ-12, is correlated with high anxiety and depression scores, as measured by the HAD Scale ($p < 0.001$).

3.1.b.3) Correlations at T_2.

A high BMI is strongly correlated with high bingeing and EDE-Q4 ($p < 0.001$) scores, and with low mastery ($p < 0.001$). A high BMI is also correlated with anxiety and depression at ($p < 0.05$). At T_2 restraint has lost its correlation with BMI ($p = 0.37$).

Cortisol secretion is no longer correlated with bingeing ($p = .78$).

High restraint remains strongly correlated ($p <.001$) with high global eating behaviour score.

High Binge score remains strongly correlated ($p < 0.001$), with high global eating behaviour score, and now with low mastery ($p < - 0.05$), and correlated with anxiety and depression ($p < 0.05$).

Low mastery is strongly correlated with high scores in global mental health pathology and anxiety and depression ($p < 0.001$), and correlated ($p < 0.05$), with global eating behaviour.

3.1.b.4) Summary of T_1 and T_2 correlations

Eating pathology as measured by the EDE-Q4 is strongly correlated with BMI throughout the study. Restraint is significant at baseline and when stress increased significantly at T_2 restraint looses its significance, with BMI. Disinhibition of restraint is coupled with a significant rise in bingeing. This rise in bingeing is significantly correlated with an increase in BMI and depression and a reduction in mastery Cortisol secretion is significantly negatively correlated with bingeing at T_1.

3.1.b.5) Bivariate correlations of change in all study variables

Bivariate correlations of change in all study variables are shown in table (3.1.5) below.

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Table 3.1.5: Bivariate correlations of change in all study variables at T_2.

	BMI Change	AUC Cortisol Change	Restraint EDE-Q4 Change	Binge EDE-Q4 Change	EDE-Q4 Global Change	Mastery Change	GHQ Change
BMI Change							
AUC cortisol Change	.46, $p < .001$**						
Restraint EDE-Q4 Change	-.78, $p < .001$**	-.58, $p < .001$**					
Binge EDE-Q4 Change	.74, $p < .001$**	.44, $p < .001$**	-.59, $p = .001$**				
EDE-Q4 Global Change	-.20, $p = .09$	-.07, $p = .55$	.50, $p = .001$**	-.10, $p = .40$			
Mastery Change	-.21, $p < .08$	-.07, $p = .53$	.24, $p < .05$*	-.13, $p = .28$	.01, $p = .96$		
GHQ Change	.01, $p = .94$	-.05, $p = .67$	.02, $p = .89$	.14, $p = .26$	.01, $p = .98$	-.28, $p < .05$*	
HAD Total Change	.06, $p = .60$	.12, $p = .34$	-.13, $p = .29$	.27, $p < .05$*	-.09, $p = .47$	-.17, $p = .15$	.50, $p < .001$**

*. *Correlation is significant at the 0.05 level (2 tailed).*

**. Correlation is significant at the 0.01 level (2 tailed).

3.1.b.6) Summary of bivariate correlations of change in all study variables at T_2

An increase in body weight is strongly associated with an increase in cortisol secretion and bingeing behaviour. These changes occur in the presence of a reduction in dietary restraint. These changes are not observed in those individuals whose body weight remained the same or decreased.

An increase in cortisol secretion is strongly associated with an increase in bingeing behaviour and a concomitant reduction in restraint.

A reduction in restraint is strongly associated with a decrease in global eating behaviour pathology, a decrease in mastery and an increase in bingeing behaviour.

Low mastery is associated with an increase in global psychiatric symptoms, particularly anxiety and depression.

3.1.c) Summary of normative data

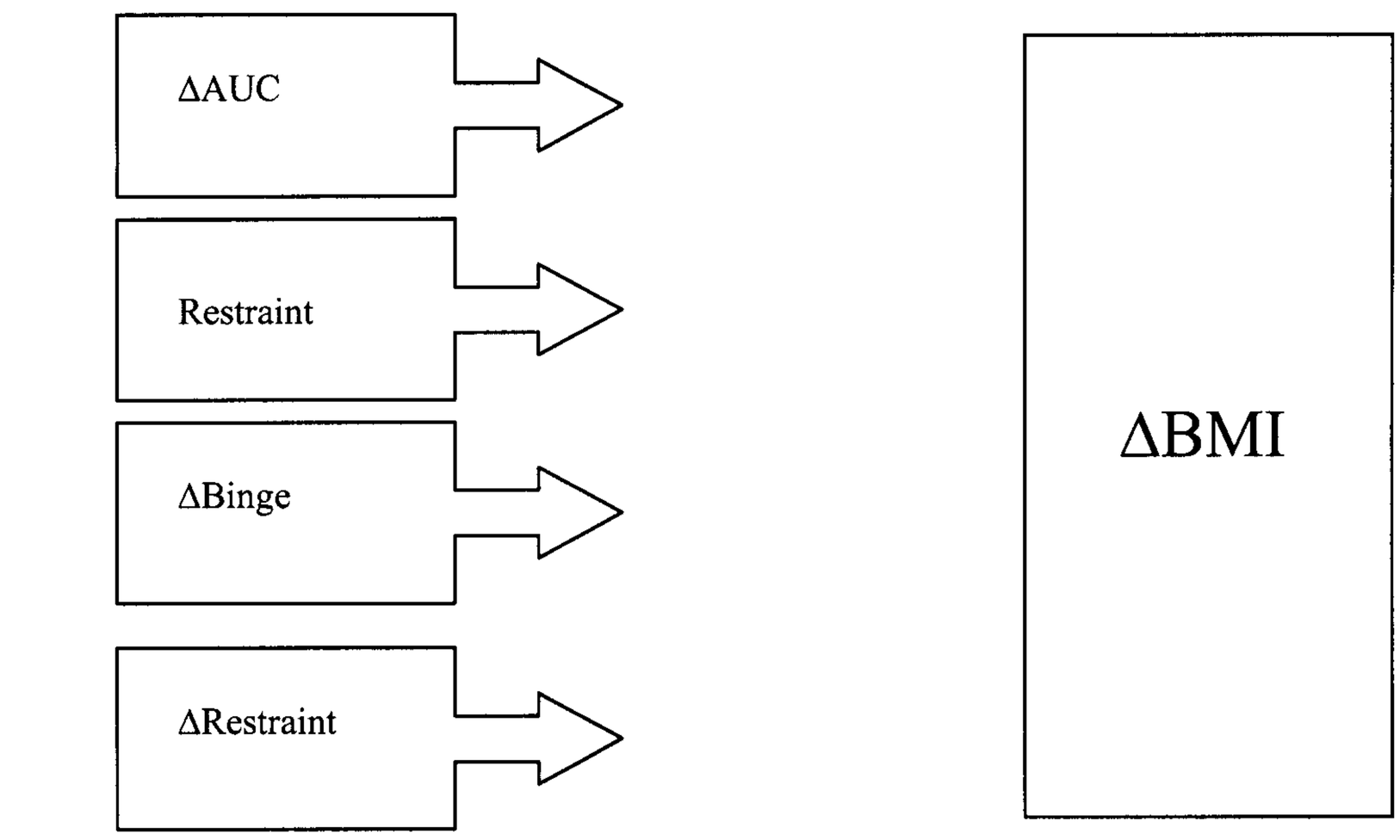
The change in BMI may be due to

- The increase in cortisol secretion
- The increase in bingeing
- The level of restraint and the subsequent decrease in restraint in the stress condition

In section 3.2 a series of linear regression analyses will explore the predictive effects of the main variables on ΔBMI (as depicted in figure 3.1.1 below). Main and

moderating effects of the potential predictive variables are presented in a series of tables.

Figure 3.1.1. Potential predictor variables which may have direct main effects on the dependent variable ΔBMI.



In accord with our hypotheses, ΔAUC cortisol, restraint, Δrestraint and ΔBingeing are potential predictive variables involved in the ΔBMI and will be explored in the next section using multiple linear regressions. These predictor variables may have direct main effects or mediate the effect of each other on ΔBMI change. For example, ΔBinge eating may mediate the effects of Δrestraint on ΔBMI. Baron and Kenny's (1986) model (as described in the Methods section). Variables that were not correlated will be left out of the model.

3.1.d) Series of regression analysis

The series of regression analysis below are presented in section 3.2.

Predicting ΔBMI: Main and moderating effects of stress and restraint.

Predicting ΔBinge: Main and moderating effects of stress and restraint.

Predicting ΔBMI: Main and moderating effects of stress and restraint, with potential mediator ΔBinge.

Predicting ΔRestraint: Main and moderating effects of stress, restraint T_1 and ΔBinge.

Predicting ΔBMI: Main and moderating effects of stress, restraint T_1, ΔBinge and Δrestraint.

Section 3.2.

In section 3.1 significant correlations were identified which suggest that ΔAUC, restraint, Δrestraint and Δbinge eating are potential predictors of change in ΔBMI.

The following linear regression analyses will explore the predictive effects of the main variables on ΔBMI. Main and moderating effects of the potential predictive variables will be presented in a series of tables.

3.2.a) Predicting ΔBMI: Main and moderating effects of stress and restraint.

As depicted in Fig 3.1.1 above regression analysis was performed to test for predictive effects of ΔAUC, restraint (T_1), and the product of restraint X ΔAUC, on the dependent variable ΔBMI.

3.2.a.1) Correlations of the predictive variables with ΔBMI.

ΔAUC ($r = 0.46$, $p < 0.001$) and restraint ($r = 0.57$, $p < 0.001$) show large correlations with ΔBMI, which are highly significant.

Product of restraint X ΔAUC ($r = 0.29$, $p < 0.01$) shows a smaller correlation, despite the significance of this correlation the coefficient is small.

Table 3.2.1: The step 1 and step 2 βweights, R^2 and ΔR^2 values for the predictive variables on the dependent variable ΔBMI.

	Step 1 (β weights)	Step 2 (β weights)
ΔAUC	.33***	.28**
Restraint T_1	.49***	.48***
Restraint T_1 x ΔAUC Product		.13
R^2	.43	.44
ΔR^2	.43	.02
F	25.53***	17.84***
d.f.	2,68	1,67

* p <.05
** p <.01
*** p <.001

From Table 4.

Step1. The R^2 value indicates that restraint and ΔAUC account for 43.0% of the ΔBMI occurring at T_2 and that this is significant.

Step 2. When the product of restraint X ΔAUC is added, there is only an increase in R^2 of 2.0%, which is not significant. The significance of ΔAUC is also reduced with restraint maintaining much the same significance.

The predictors restraint (T_1) and ΔAUC show a significant main effect on the increase in BMI (ΔBMI),. There is no significant interaction between restraint and ΔAUC on ΔBMI at T_2.

This data confirms ΔAUC and restraint as significant predictors in ΔBMI with no moderating effect in either variable.

3.2.a.2) Summary of the predictive effects of ΔAUC and restraint T_1 on ΔBMI

Both restraint and the increase in cortisol secretion have been shown to be significant predictors of ΔBMI. (see figure 3.2.1 below). The next regression analysis is the second in the series of three analyses. The second regression analysis will explore the effect of these predictors on binge change in order to perform analysis 3, which will enable us to identify if binge eating (ΔBinge), is a mediator of the predictors ΔAUC and restraint on ΔBMI.

Figure 3.2.1: Model showing significant predictor variables on the dependent variable change in BMI.

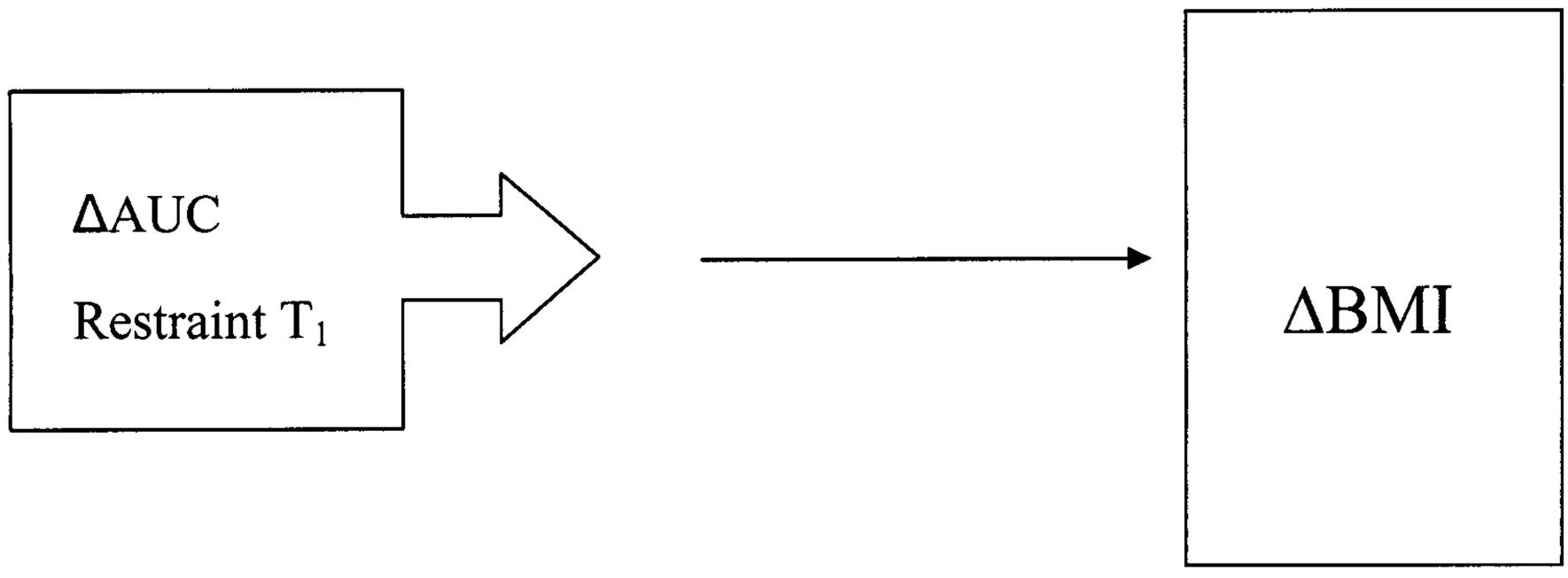
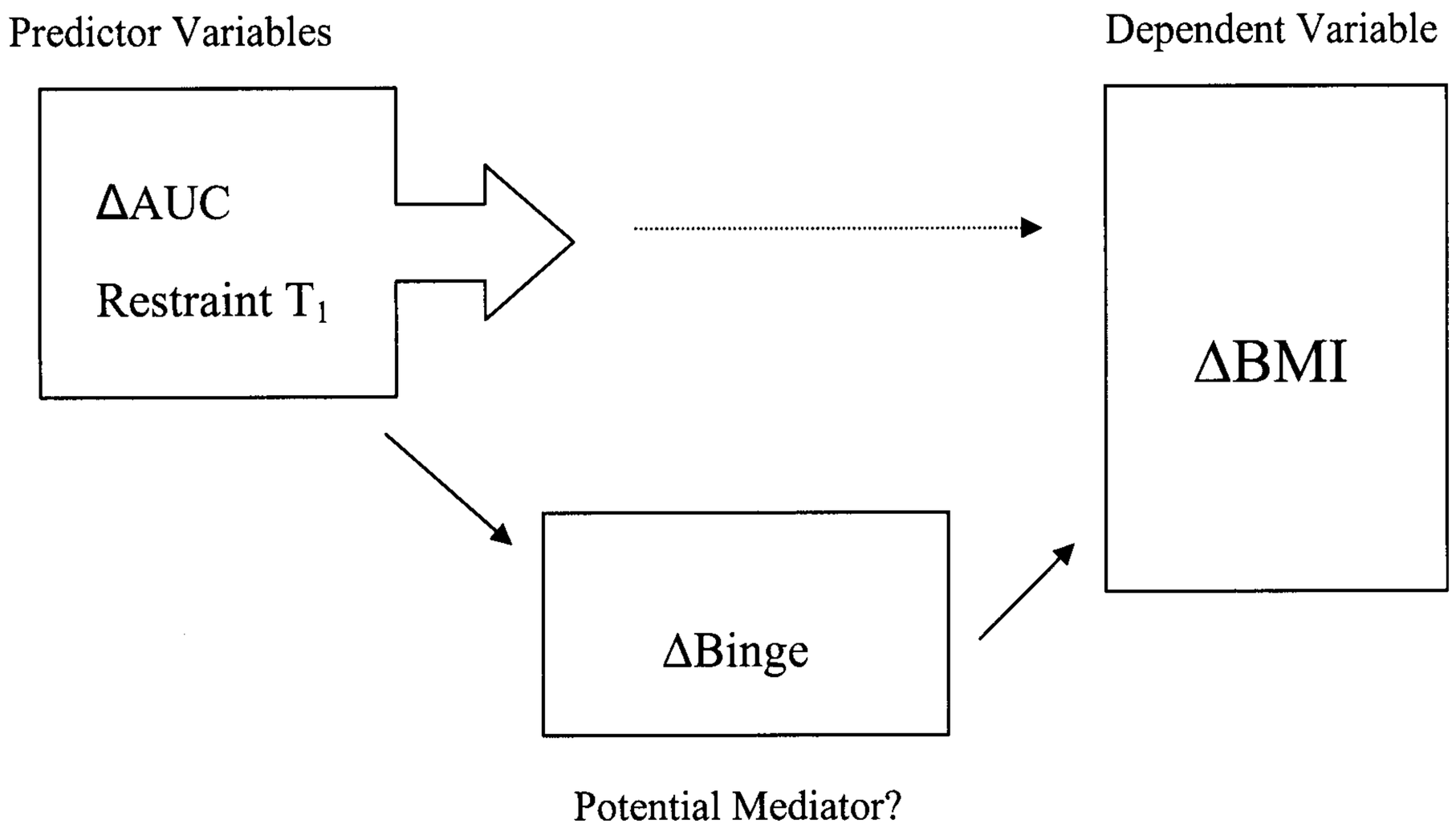


Figure 3.2.2: *Expected* effects of the predictor variables on the dependent variable, may be mediated by ΔBinge.



3.2.b) Predicting ΔBinge: Main and moderating effects of stress and restraint.

The next regression analysis was performed to test for predictive effects of ΔAUC, restraint (T_1), and the product of restraint X ΔAUC, on the dependent variable ΔBinge. This data is required to explore any mediating effects the change in binge may have in the effects of stress and restraint on body weight change.

3.2.b.1) Correlations of the predictive variables with ΔBinge.

ΔAUC (r = 0.44, $p < 0.001$), Restraint ($r = 0.56$, $p < 0.001$) and Product of Restraint X ΔAUC ($r = 0.33$, $p = 0.01$), show large positive correlations with ΔBinge, which are highly significant.

Table 3.2.2: The step 1 and step 2 βweights, R^2 and ΔR^2 values for the predictive variables on the dependent variable ΔBinge.

	Step 1 (β weights)	Step 2 (β weights)
ΔAUC	.32**	.25*
Restraint T_1	.48***	.47***
Restraint T_1 x ΔAUC Product		.19
R^2	.41	.44
ΔR^2	.41	.03
F	23.16***	17.33***
d.f.	2,68	1,67

* p <.05
** p <.01
*** p <.001

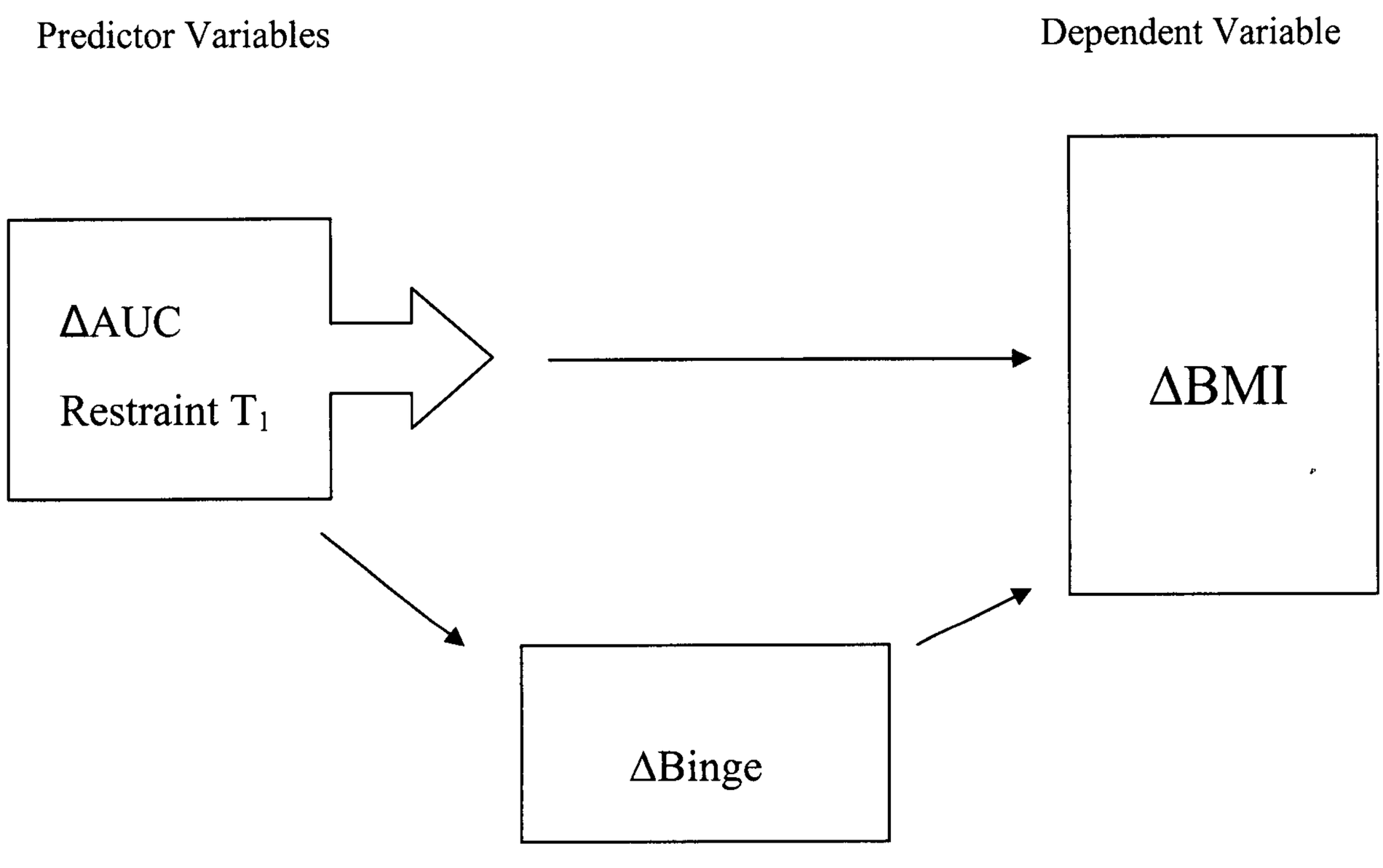
Step 1. The R^2 value indicates that restraint and ΔAUC account for 41.0% ΔBinge eating occurring at T_2 and that this is significant.

Step 2. When the product of restraint X ΔAUC is added, there is a small increase in ΔBinge which is of borderline significance $p = .056$. ΔAUC is reduced in significance, whilst the significance of restraint on ΔBinge remains the same.

3.2.b.2) Summary of the main and moderating effects of ΔAUC and restraint T_1 on ΔBinge

This indicates that change binge may be a partial mediator of the predictive effects of stress on change in body weight. Therefore a direct main effect of restraint and ΔAUC on the ΔBinge, between T_1 and T_2 is demonstrated. There is a borderline significant interaction between restraint and ΔAUC on Δ Binge at T_2 indicating a possible moderating effect.

Figure 3.2.3. *Confirmed* effects of the predictor variables on the dependent variable, may be partially mediated by ΔBinge.



Regression analysis 3 is required to test for a mediating effect of ΔBinge in the effects of ΔAUC, restraint and the product of restraint X ΔAUC on ΔBMI.

Regression analysis 2 confirms that ΔAUC and restraint are predictors of ΔBinge. The next step is regression analysis 3, which if our hypothesis is correct will confirm that ΔBinge is a mediator between the predictors ΔAUC and restraint.

3.2.c) Predicting ΔBMI: Main and moderating effects of stress and restraint, with potential mediator ΔBinge

This regression analysis was performed to test for predictive effects of ΔAUC, restraint (T_1), and potential mediating role of ΔBinge, on the dependent variable ΔBMI.

3.2.c.1) Correlations of the predictive variables with ΔBMI.

ΔAUC ($r = 0.46$, $p = 0.001$) and restraint T_1 ($r = 0.57$, $p = 0.001$) show large correlations with ΔBMI, which are highly significant.

ΔBinge ($r = 0.74$, $p = 0.001$) shows the largest correlation of the three predictors and is highly significant. This may indicate a potential role as a mediator of the direct main effects, of ΔAUC and restraint T_1 on the dependent variable ΔBMI.

Table 3.2.3: The step 1 and step 2 βweights, R^2 and ΔR^2 values for the predictive variables on the dependent variable ΔBMI.

	Step 1 (β weights)	Step 2 (β weights)
ΔAUC	.33***	.16
Restraint T_1	.49***	.23*
ΔBinge		.55***
R^2	.43	.61
ΔR^2	.43	.18
F	25.53***	30.41***
d.f.	2,68	1,67

* p <.05
** p <.01
*** p <.001

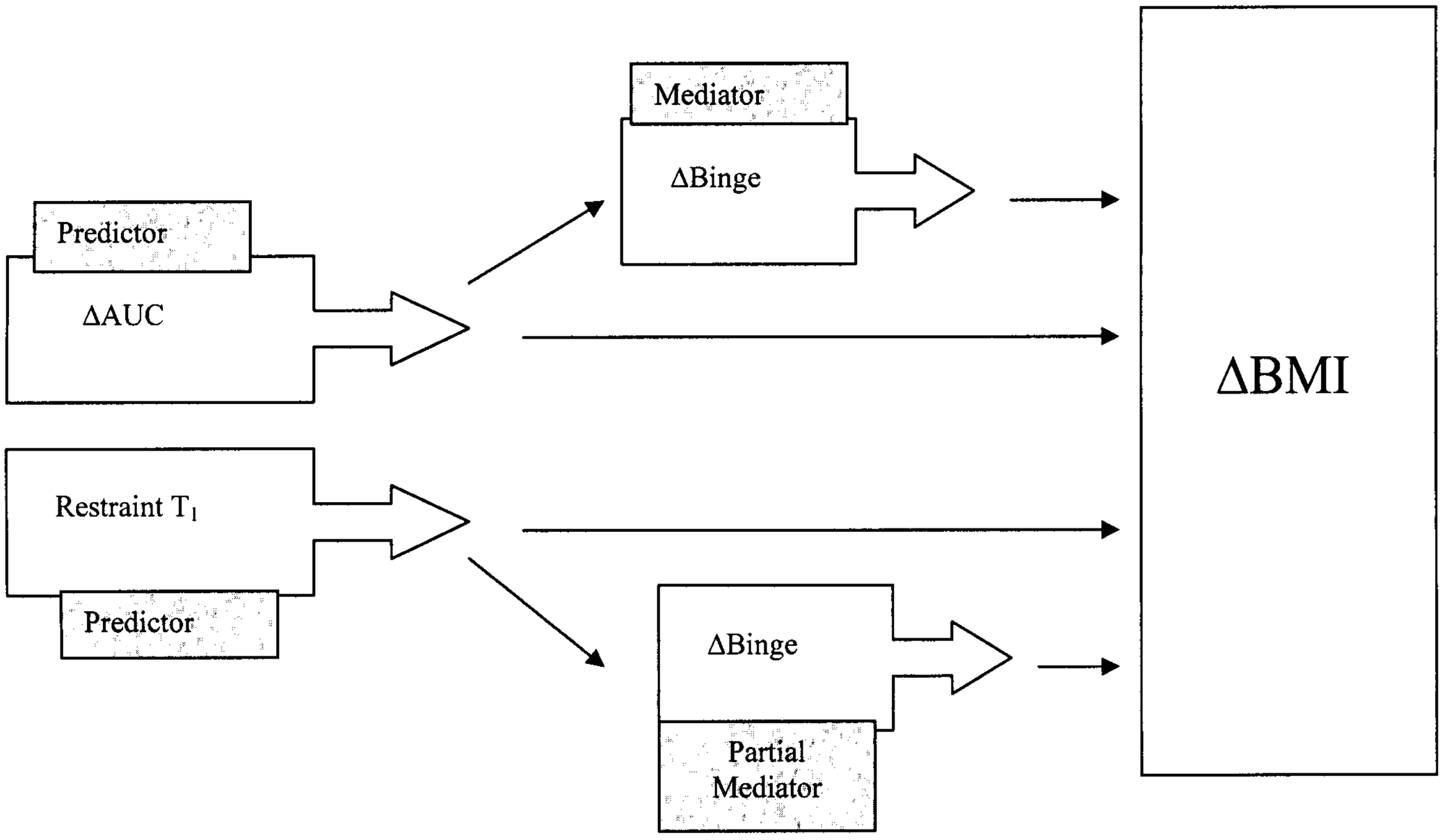
Step 1. The R^2 value indicates that restraint T_1 and ΔAUC account for 43.0% of the change in ΔBMI occurring at T_2 and that this is highly significant. Therefore, there is a direct main effect of ΔAUC, restraint T_1 on ΔBMI.

Step 2. When ΔBinge is added, there is an increase in the amount of variance accounted for by 18% to a total of 61%, this is highly significant. The effect of ΔAUC is reduced to insignificance, whilst restraint T_1 changes from being highly significant to being significant ($p < .05$).

3.2.c.2) Summary of the main and moderating effects of stress and restraint, with potential mediator ΔBinge on ΔBMI

This indicates that ΔBinge is a mediator of the effects of the predictor ΔAUC T_1 on the criterion variable ΔBMI. The reduction in significance of restraint T_1 may indicate a role as a partial mediator of ΔBMI.

Figure 3.2.4: Model showing ΔAUC and restraint T_1 are predictors of ΔBMI. ΔBinge mediates the predictive effects of ΔAUC and partially mediates the effects of restraint T_1 on ΔBMI



This series of three regression analyses have shown that ΔAUC and restraint T_1 have direct main effects on ΔBMI, and that ΔBinge is a mediator of the predictive effects of ΔAUC and a partial mediator of the predictive effects of restraint on ΔBMI

Cortisol is known to increase hunger and food intake in chronic stress therefore providing energy for physiological responses to stress. So in general binge eating emerges as a significant mediator of the effects of stress on ΔBMI.

Chapter 4 will explore differences in those who gained weight, lost weight and those who remained the same weight.

In the previous regression analysis, it was shown that ΔBinge is a mediator of the predictive effects of ΔAUC and a partial mediator of the effects of restraint T_1 on ΔBMI. The question arises as to what other potential predictors and mediators play a role in ΔBMI. There is a significant reduction in restraint at T_2 as compared with T_1. Accordingly, the next regression analysis will explore the role of ΔRestraint, if any on the dependent variable ΔBMI, and ΔRestraint's potential mediating role in the effects of predictors on ΔBMI.

3.2.d) Predicting ΔRestraint: Main and moderating effects of stress, restraint T_1 and ΔBinge.

This regression analysis was performed to test for predictive effects of ΔAUC, restraint (T_1), and the product of restraint X ΔAUC, on ΔRestraint.

3.2.d.1) Correlations of the predictive variables with ΔRestraint.

ΔAUC (r = -.58, p = 0.001) restraint T_1 (r = -.56, p = 0.001) and product of restraint X ΔAUC (r = -.42, p = 0.001) show large negative correlations with ΔRestraint, which are highly significant.

Table 3.2.4: The step 1 and step 2 β weights, R^2 and ΔR^2 values for the predictive variables on the dependent variable ΔRetraint.

	Step 1 (β weights)	Step 2 (β weights)
ΔAUC	-.47***	-.40***
Restraint T_1	-.44***	-.43***
Restraint T_1 x ΔAUC Product		-.21*
R^2	.52	.56
ΔR^2	.52	.04
F	36.40***	27.91*
d.f.	2,68	1,67

* p <.05
** p <.01
*** p <.001

Step 1. The R^2 value indicates that restraint and ΔAUC account for 52.0% of the ΔRestraint occurring at T_2 and that this is highly significant.

Step 2. When the product of restraint T_1 X ΔAUC is added, there is an increase in R^2 4.0%, which is significant p<.05. Restraint (T_1) and ΔAUC remain highly significant.

The predictors Restraint (T_1) and ΔAUC show a highly significant main effect on the ΔRestraint in both steps. There is a significant interaction between Restraint (T_1) X ΔAUC on ΔRestraint.

3.2.d.2) Summary of the main and moderating effects of stress, restraint T_1 and ΔBinge on ΔRestraint

This data confirms ΔAUC and Restraint (T_1) as significant predictors in ΔRestraint with a significant moderating effect.

The next regression analysis will explore any potential mediating role ΔRestraint may play in the effects of these mediators on ΔBMI

3.2.e) Predicting ΔBMI: Main and moderating effects of stress, restraint T_1, ΔBinge and ΔRestraint.

This regression analysis was performed to test for predictive effects of ΔAUC, restraint (T_1), ΔBinge and ΔRestraint, on the dependent variable ΔBMI.

3.2.e.1) Correlations of the predictive variables with ΔBMI.

ΔAUC ($r = 0.45$, $p = 0.001$), restraint T_1 ($r = 0.57$, $p = 0.001$), and ΔBinge ($r = 0.74$, $p = 0.001$), show large positive correlations with ΔBMI, which are highly significant. ΔRestraint ($r = -.78$, $p = 0.001$) shows a very large negative correlation with ΔBMI, which is also highly significant.

Table 3.2.5: The step 1 and step 2 β weights, R^2 and ΔR^2 values for the predictive variables on the dependent variable ΔBMI.

	Step 1 (β weights)	Step 2 (β weights)
ΔAUC	.33***	-.056
Restraint T_1	.49***	.052
ΔBinge		.42***
ΔRestraint		-.53***
R^2	.43	.74
ΔR^2	.43	.31
F	25.35***	42.23***
d.f.	2,68	1,67

* p <.05
** p <.01
*** p <.001

Step 1. The R^2 value indicates that restraint and ΔAUC account for 43.0% of the ΔBMI occurring at T_2 and that this is highly significant.

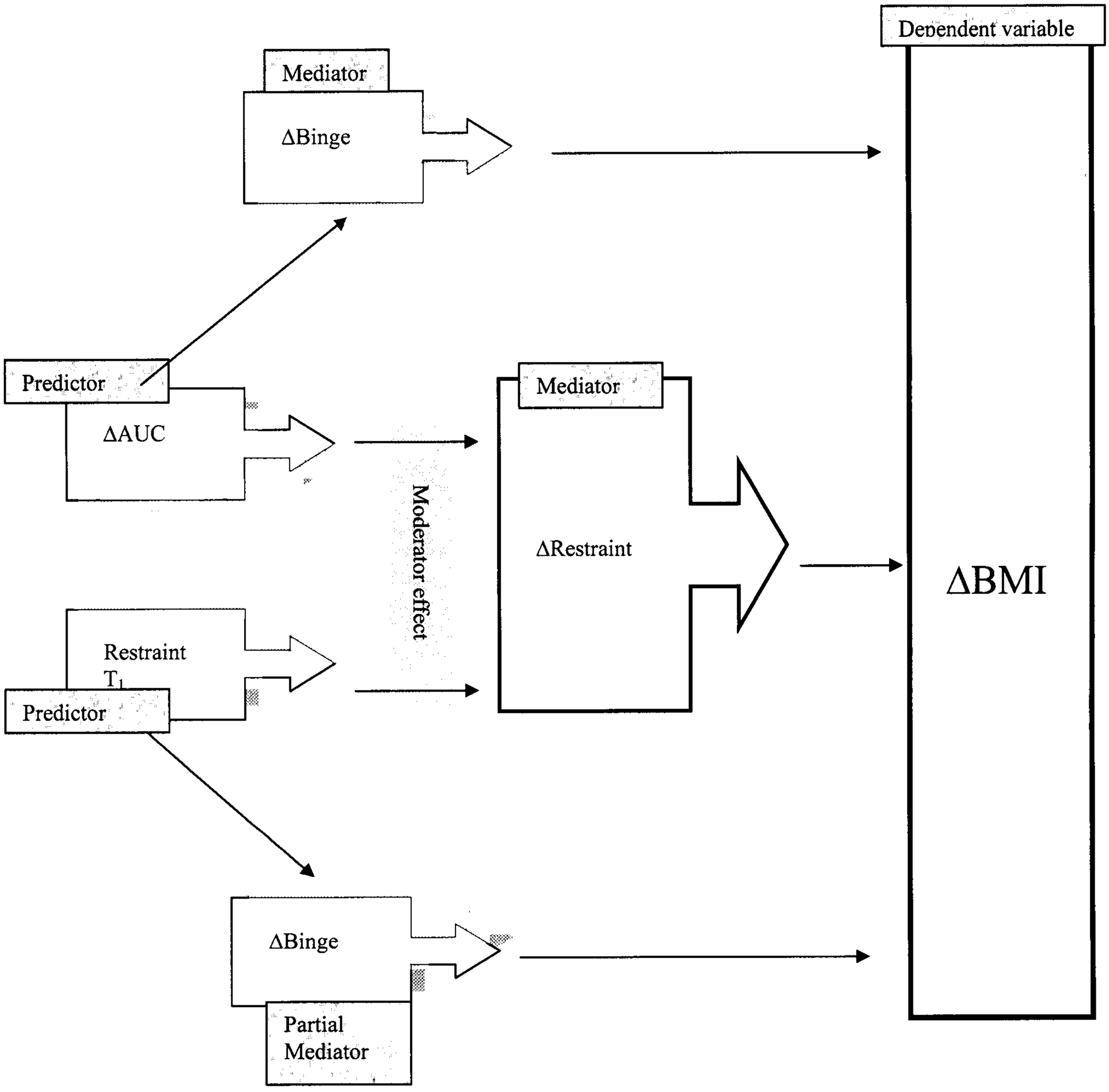
Step 2. When ΔBinge and ΔRestraint are added the R^2 value indicates a large increase of 31% that is highly significant, inducing an overall change of 74% on the dependent variable ΔBMI. When ΔRestraint is added into the model regression model ΔAUC and restraint T_1 become insignificant. This confirms that ΔRestraint is a mediator of the predictive variables ΔAUC and Restraint T_1on ΔBMI.

3.2.e.2) Summary of the main and moderating effects of stress, restraint T_1, ΔBinge and Δrestraint on ΔBMI

This data confirms ΔAUC, Restraint T_1, ΔBinge and ΔRestraint as significant predictors in ΔBMI.

Figure 3.2.5: The relationship between predictors, mediators and the dependent variable ΔBMI.

The previous regression analyses provide support for the model shown in this figure.



In the next section a series of three regression analyses (shown below), will be presented from a sub group of individuals n = 38. This will enable the variable FFQ to be evaluated in this group. Total calorific intake and food choice will be evaluated at base line T_1 and the stress period T_2.

Predicting ΔBMI: Main and moderating effects of stress and restraint.

Predicting ΔFFQ: Main and moderating effects of stress and restraint.

Predicting ΔBMI: Main and moderating effects of stress, restraint and food choice.

Section 3.3

3.3.a) Introduction to the Food Frequency Questionnaire (FFQ), used in a sub group of subjects.

A sub group of 38 individuals completed The Food Frequency Questionnaire. This questionnaire was added to the study to explore food choice and quantity of food consumed. For example, in the stress condition, does the carbohydrate and saturated fat intake increase? Significant correlations, T-tests and regression analysis will be presented as in the previous sections of Chapter 3.

3.3.a.1) Food Frequency Questionnaire Descriptive statistics.

Table 3.3.1: Table to show Food Frequency Questionnaire (FFQ), descriptive statistics.

	T_1 MEAN (SD) n = 38	T_2 MEAN (SD) n = 38	t
ALKCAL	1529.67 (387.74)	1877.92 (530.13)	-4.10***
KCAL	1488.06 (378.28)	1822.09 (513.98)	-4.09***
ALC ONLY	41.61 (29.45))	55.83 (37.60)	-3.15**
FAT	58.17 (19.31)	72.53 (22.09)	-4.18***
POLFAT	11.12 (3.85)	11.95 (3.65)	-1.91
SATFAT	22.61 (8.45)	28.76 (9.62)	-4.46***
CHO	187.36 (51.05)	240.44 (79.33)	-4.30***
PROTEIN	65.73 (15.65)	67.73 (16.56)	-0.89
SUGAR	86.09 (26.45)	112.05 (40.89)	-4.11**
STARCH	100.70 (32.82)	127.22 (42.84)	-4.00**
PERPROT	17.86 (2.14)	15.23 (2.56)	4.88***
PERFAT	34.79 (5.65)	35.81 (4.71)	-2.10*
PERCHO	47.40 (6.27)	49.14 (5.55)	-2.62*
PSRATIO	0.53 (0.17)	0.45 (0.18)	3.70***

Key:
ALKCAL = total dietary kcalories inclusive of alcohol.

KCAL = total dietary kcalories exclusive of alcohol.

POLFAT = polyunsaturated fat.

PERFAT = percentage fat.

PSRATIO = polyunsaturated fat to saturated fat ratio.

The negative t values indicate an increase in these variables at T_2. Polyunsaturated fat and protein are the only dietary variables which didn't increase significantly at T_2.

Table 3.3.2: Mean and standard deviations of main study variables in the whole group n = 71 vs the subgroup completing the FFQ n = 38.

	Mean (s.d.) n = 38	Mean (s.d.) n = 71
ΔBMI	0.35 (.55)	0.31 (0.61
ΔAUC	14.01 (18.37)	15.50 (21.20)
Restraint T_1	1.87 (1.26)	1.77 (1.29)
Restraint x ΔAUC	0.38 (0.93)	0.26 (1.17)

Regression analysis will explore the role of food quantity and choice as a factor of binge eating that may account for a change in BMI. Regression analysis will also be used to explore ΔBMI and main and moderating effects of stress and restraint in this subgroup n = 38. If we can show mediating and moderator effects of variables in the sub group follow similar relationships to the whole group then we can suggest the FFQ findings are equivalent to the whole group.

Correlations within the subgroup (n = 38), follow very similar patterns to those in the whole group (n = 71).

When FFQ is included a significant correlation is seen between bingeing and FFQ at T_2 ($p < .01$). This would suggest that food quantity and choice have a significant effect on ΔBMI. The following statistical analysis addresses this hypothesis.

3.3.b) Correlations for FFQ and Binge T_1 & T_2

T_1 ($r = -0.11, p = .52$)

T_2 ($r = 0.43, p < .01$)

The following regression analyses, were conducted to test the hypothesis that food quantity and choice would have a significant effect on ΔBMI.

3.3.c) Predicting ΔBMI: Main and moderating effects of stress and restraint. n = 38.

3.3.c.1) Correlations of the predictive variables with ΔBMI.

ΔAUC ($r = 0.60, p < 0.001$) and Restraint T_1 ($r = 0.61, p < 0.001$) show large correlations with ΔBMI, which are highly significant. In this group of 38 individuals the correlation is greater than in the main group n = 71.

Product of Restraint X ΔAUC ($r = 0.10, p = 0.22$) shows a smaller correlation, which is not significant.

Table 3.3.3: The step 1 and step 2 β weights, R^2 and ΔR^2 values for the predictive variables on the dependent variable ΔBMI.

	Step 1 (β weights)	Step 2 (β weights)
ΔAUC	.41**	.50**
Restraint T_1	.44***	.41***
Restraint x ΔAUC Product		-.16
R^2	.51	.53
ΔR^2	.51	.02
F	18.12	12.67
d.f.	2,35	1,34

* p <.05
** p <.01
*** p <.001

Step 1. The R^2 value indicates that restraint T_1 and ΔAUC account for 51.0% of the ΔBMI occurring at T_2 and that this is significant.

Step 2. When the product of restraint X ΔAUC is added there is only an increase in R^2 2.0%. Which is not significant.

There is no significant interaction between restraint and ΔAUC on ΔBMI shown by the non-significant β weight.

In this group N = 38, the data confirms ΔAUC and restraint as significant predictors in ΔBMI with no moderating effect in either variable.

3.3.c.2) Summary of the main and moderating effects of stress and restraint on ΔBMI.

Regression analysis was conducted in this group of individuals who completed the

FFQ (n = 38), to establish ΔAUC and restraint as significant predictors in ΔBMI.

ΔAUC and restraint have the same predictive effect on the ΔBMI as in the total group n = 71, and that there is no moderating effect between the two predictors.

The next regression analysis will be performed for any observable predictive effects of ΔAUC and restraint T_1 on ΔFFQ.

3.3.d) Predicting ΔFFQ: Main and moderating effects of stress and restraint.

This regression analysis was performed to test for predictive effects of ΔAUC, restraint T_1, and the product of restraint X ΔAUC, on change in Food Factor Questionnaire (ΔFFQ). The ΔFFQ is equivalent to the change in total calorific intake including alcohol.

3.3.d.1) Correlations of the predictive variables with ΔFFQ.

ΔAUC ($r = 0.49$, $p < 0.005$) and Restraint T_1 ($r = 0.34$, $p < 0.05$) show large correlations with ΔFFQ. ΔAUC is highly significant.

Product of Restraint X ΔAUC ($r = 0.24$, $p = 0.072$) shows a smaller correlation; which is not significant.

Table 3.3.4: The step 1 and step 2 β weights, R^2 and ΔR^2 values for the predictive variables on the dependent variable ΔFFQ.

	Step 1 (β weights)	Step 2 (β weights)
ΔAUC	.42*	.40*
Restraint T_1	.16	.16
Restraint x ΔAUC Product		.02
R^2	.26	.26
ΔR^2	.26	.00
F	7.04**	4.77*
d.f.	2,35	1,34

* p <.05
** p <.01
*** p <.001

Step 1. The R^2 value indicates that restraint and ΔAUC account for 26.0% of the ΔFFQ occurring at T_2 and that this is significant.

Step 2. When the product of restraint X ΔAUC is added there is no increase in effect as measured by R^2 .

The predictors restraint (T_1) and ΔAUC show a significant main effect on the increase in FFQ (ΔFFQ). There is no significant interaction between restraint and ΔAUC on ΔFFQ at T_2.

3.3.d.2) Summary of the main and moderating effects of stress and restraint ΔFFQ.

The regression analysis exploring the effects of the predictors ΔAUC and restraint T_1 has shown that ΔAUC has a significant direct main effect in the dependent variable ΔFFQ, whilst restraint T_1 has no effect.

3.3.e) Predicting ΔBMI: Main and moderating effects of stress, restraint and food choice

This regression analysis was performed to test for predictive effects of ΔAUC, restraint (T_1), and the ΔFFQ, on the dependent variable ΔBMI. The ΔFFQ is equivalent to the change in total calorific intake including alcohol.

3.3.e.1) Correlations of the predictive variables with ΔBMI.

ΔAUC (r= 0.60, $p < 0.001$), Restraint ($r = 0.61$, $p < 0.001$) and ΔFFQ ($r = 0.58$, $p =$ 0.001) show large correlations with ΔBMI, which are highly significant.

Table 3.3.5: The step 1 and step 2 β weights, R^2 and ΔR^2 values for the predictive variables on the dependent variable ΔBMI.

	Step 1 (β weights)	Step 2 (β weights)
ΔAUC	.41**	.27*
Restraint T_1	.44**	.38**
ALKCALCH		.31*
R^2	.51	.59
ΔR^2	.51	.08
F	18.12***	15.9***
d.f.	2,35	1,34

* p <.05
** p <.01
*** p <.001

Step 1. The R^2 value indicates that restraint and ΔAUC account for 51.0% of the ΔBMI occurring at T_2 and that this is highly significant.

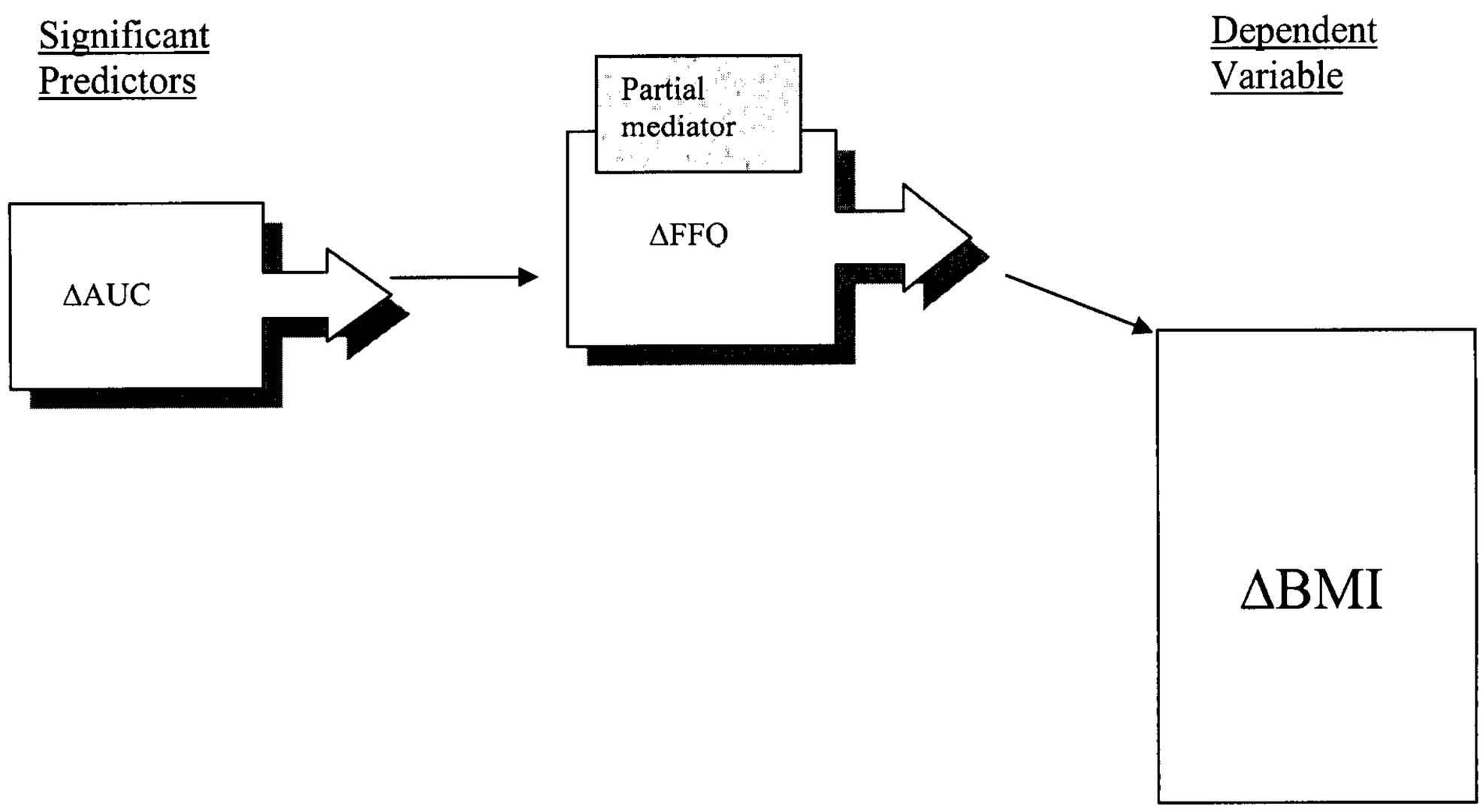
Step 2. When ΔFFQ is added there is an increase in R^2 8.0%, to a total of 59%, which is significant. The reduction in significance of ΔAUC may indicate that ΔFFQ partially mediates the effect of the predictor ΔAUC on the dependent variable ΔBMI.

3.3.e.2) Summary of the main and moderating effects of stress, restraint and food choice on ΔBMI

The results of this regression analysis indicate there is a direct main effect of ΔAUC, restraint and ΔFFQ on ΔBMI, between T_1 and T_2.

The model below is supported by the regression analyses explored in this chapter. A summary follows and should be read with the model below.

Fig 3.3.1: Model supported by regression analyses above. It shows the partial mediating role of ΔFFQ on the predictive effects of ΔAUC on ΔBMI.



3.3.f) List and overview of regression analysis.

N=71
Predicting ΔBMI: Main and moderating effects of stress and restraint; shows ΔAUC and Restraint T_1 are predictors of ΔBMI. In fact they account for 43 % of the change in BMI. The ΔAUC clearly shows the study individuals were more highly stressed at T_2 compared to T_1. The question that arises is what accounts for the rest of the change in BMI?

Predicting ΔBinge: Main and moderating effects of stress and restraint; shows that ΔAUC and Restraint T_1 account for 41% of the change in bingeing behaviour.

Predicting ΔBMI: Main and moderating effects of stress and restraint, with potential mediator ΔBinge; shows that ΔBinge mediates the effects of ΔAUC on ΔBMI. The change in bingeing is also a partial mediator of the effects of Restraint T_1 on ΔBMI.

Predicting ΔRestraint: Main and moderating effects of stress, restraint T_1 and ΔBinge;.indicates ΔAUC and Restraint T_1 are highly significant predictors of ΔRestraint. ΔAUC and Restraint T_1 are also significant moderators of Δrestraint.

Predicting ΔBMI: Main and moderating effects of stress, restraint T_1, ΔBinge and ΔRestraint; indicates that ΔRestraint mediates the effects of the predictors ΔAUC and Restraint T_1 on the dependent variable ΔBMI.

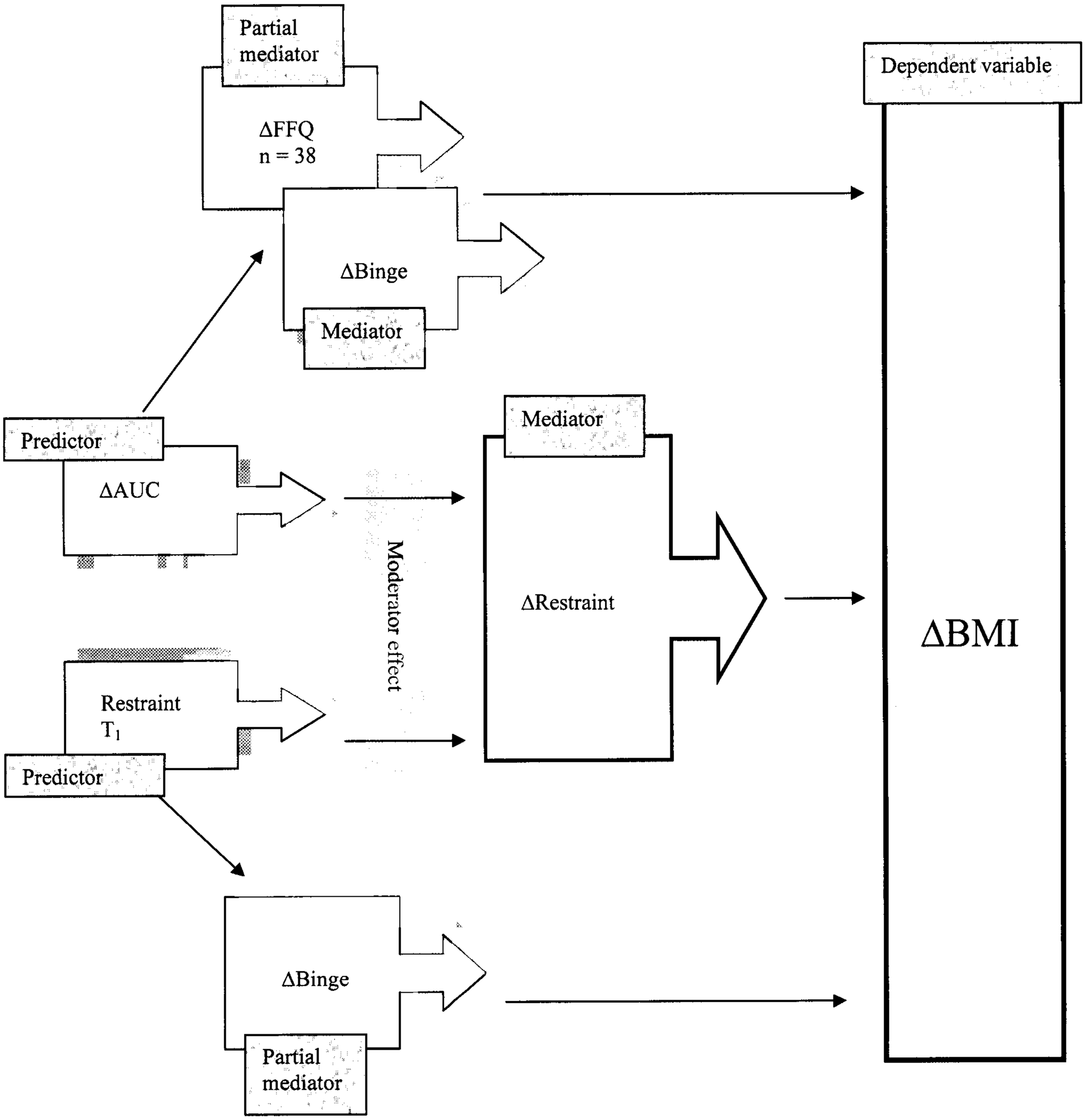
Regression analyses 4,5 and 6 were conducted on a subgroup of individuals n = 38 to test the hypothesis that food quantity and choice would be significant predictors of ΔBMI.

N = 38
Predicting ΔBMI: Main and moderating effects of stress and restraint; shows that ΔAUC and Restraint T_1 are predictors of ΔBMI. In fact they account for 51 % of the change in BMI in this sub group.

Predicting ΔFFQ: Main and moderating effects of stress and restraint; shows that ΔAUC and Restraint T_1 are predictors of ΔFFQ. In fact they account for 26 % of the change in FFQ in this sub group.

Predicting ΔBMI: Main and moderating effects of stress, restraint and food choice; shows that ΔFFQ is a partial mediator of the predictor AUC on change in BMI. This is shown by a reduction of the effects of ΔAUC on ΔBMI, which remains within the 5% significance level $p < 0.05$.

Figure 3.3.2. Model supported by the preceding regression analysis to explain the relationship between predictors, mediators, partial mediators and the dependent variable ΔBMI.



Predictors ΔAUC, Restraint T1
Mediators/ partial mediators ΔFFQ, ΔBinge and ΔRestraint,
Dependent variable ΔBMI.
The analysis of the partial mediator role of ΔFFQ was conducted on a subgroup of 38 individuals. All other analysis shown was conducted on data from the whole group (n = 71).

3.3.f.1) Summary of Chapter three.

This series of regression analyses provides support for the model in fig 3.3.2 above. In this longitudinal study an academic exam was a significant stressor and the increase in salivary cortisol, confirms this. Weight had increased significantly by the time of the exam. Significant predictors of weight change were found to be stress, dietary restraint, inhibition of dietary restraint, bingeing and food choice. Individuals who were high in dietary restraint at the beginning of the study became unrestrained during the stress period. This change in restraint was found to impact significantly on the increase in body weight. The change in restraint acted as a mediator and moderator of the effects of stress and baseline restraint score on the change in body weight. During the stress period, bingeing behaviour increased and was found to be a significant mediator of the effects of stress on the increase in body weight. An increase in the consumption of saturated fat, carbohydrates and alcohol and a decrease in protein intake were reported during the stress period. This change in food consumption partially mediated the effects of stress on body weight. This supports the "comfort-eating" hypothesis, i.e. the consumption of high carbohydrate, and saturated fatty foods in an attempt to alleviate or avoid the psychological consequences of stress. A decrease in coping was evident, as mastery decreased and bingeing increased during the stress period. Throughout this time, depression increased and anxiety remained much the same as base line levels. Depression in the group as a whole increased to a level of "caseness" indicating significant levels of depression during the stress period.

The next chapter will explore descriptive statistics and compare variables at baseline with any changes during the stress period in three subgroups identified within the main group n = 71. This information may identify individual attributes that are likely to increase weight, lose weight or maintain weight. Regression analysis is incumbent within the main regression analysis in Chapter 3. The cohort consisted of 71 participants and their measurements are shown in Table 3.1.1 on page 100).

Chapter 4

Results

This chapter will be divided into two sections;

Section 1. Comparison of subjects who changed and those who remained the same weight.

Section 2. Investigation of a return to baseline in subjects completing T_3.

Section 1.

In this section the main study variables will be compared on the three groups within the 71 subjects.

The three groups are those who;

1. Increased BMI (n = 40)
2. Decreased BMI (n = 19)
3. BMI unchanged (n = 12).

The purpose of Chapter 4, section 1, is to explore any defining attributes within the three groups that might enable identification of individuals that are likely to change weight in response to stress.

4.1) Introduction: comparison of the main study variables in the three sub groups.

In the last chapter regression analysis confirmed change in cortisol and dietary restraint moderate the change in BMI during the stress period. In addition, change in restraint is a significant complete mediator of change in cortisol secretion and restraint at baseline on weight change. Binge eating and a change in food choice, for example an increase in CHO and saturated fat, were also predictive of changes in weight and mediators of restraint and change in cortisol on weight change. (See fig 3.3.2).

The aim of this chapter is to explore descriptive statistics and compare variables at base line with any changes during the stress period in the three subgroups. This information may be useful in identifying any attributes individuals may have who are likely to increase weight (n = 40), loose weight (n = 19), or remain the same weight (n = 12). Regression analysis is incumbent within the main regression analysis discussed in Chapter 3. The cohort consisted of 71 participants and their measurements are shown in Table 3.1.1 page 100.

The evaluations of the groups will follow the same descriptive statistical format found in Chapter 3. In that group mean, standard deviation, T-statistic and significance will be presented.

The next section will present the main variables measured in the study for all three groups.

A discussion comparing the three groups will follow the tables presented below.

Table 4.1.1: Measurements at baseline T_1, in the whole group n = 71 and the subgroups.

	T_1 Mean (SD) N=71 Whole Group	T_1 Mean (SD) N=40 Increased wt	T_1 Mean (SD) N=19 Decreased wt	T_1 Mean (SD) N=12 Constant wt	Did not complete T_2 N = 13
Height	1.63 (0.1)	1.64 (0.07)	1.64 (0.07)	1.62 (0.08)	1.65 (0.07)
Age	43.0 (7.1)				44.16 (7.8)
AUC	77.3 (26.6)	70.0 (22.6)	89.5 (26.9)	82.9 (31.8)	58.9 (20.5)
Restraint	1.8 (1.3)	2.4 (0.9)	1.3 (1.1)	0.9 (1.2)	1.9 (1.3)
BMI	25.2 (4.3)	26.11 (4.7)	24.08 (3.2)	24.23 (3.76)	25.72 (3.6)
Binge	0.50 (0.7)	0.73 (0.8)	0.26 (0.6)	0.08 (0.3)	0.54 (0.7)
GHQ	2.6 (3.0)	2.8 (2.9)	2.63 (3.55)	1.9 (2.6)	2.2 (3.5)
HAD anx	7.5 (3.9)	8.0 (3.7)	6.7 (4.4)	7.3 (3.5)	7.8 (4.1)
HAD dep	3.9 (3.3)	4.5 (3.1)	3.1 (3.6)	3.3 (3.3)	4.5 (3.9)
HAD Total	11.4 (6.6)	12.5 (6.1)	9.8 (7.9)	10.5 (6.23)	12.3 (6.9)
LTE	0.86 (1.1)	0.75 (1.0)	1.05 (1.3)	0.92 (1.2)	1.08 (1.8)
Mastery	21.3 (3.1)	20.7 (3.1)	21.9 (3.5)	22.17 (2.6)	21.08 (1.6)
EDE-Q4 Total	2.31 (1.2)	2.6 (1.0)	1.8 (1.1)	1.6 (0.9)	2.6 (1.3)

Table 4.1.1 displays the baseline measurements for the whole group and subgroups, for comparison. Deviation from baseline in all groups will be discussed in the following sections. Variable measurements taken at T_2 will be presented alongside baseline measurements as appropriate.

4.1.a) Characteristics of the increased weight group (n = 40).

Table 4.1.2: Mean and standard deviation at T_1 and T_2 of the main predictors in the study and the dependent variable (BMI), for the 40 individuals who increased in weight.

	T_1 Mean (SD) n = 40	T_2 Mean (SD) n = 40	t	Significance (2 tailed) T_1 vs T_2
Height	1.64 (0.1)			
AUC	70.0 (22.6)	93.87 (29.3)	-8.95	0.001
Restraint	2.43 (0.9)	1.15 (0.6)	11.88	0.001
BMI	26.11 (4.7)	26.87 (4.8)	-13.80	0.001
Binge	0.73 (0.8)	2.25 (2.0)	-5.28	0.001
GHQ	2.80 (2.9)	4.57 (3.3)	-3.58	0.01
HAD anx	8.0 (3.7)	9.5 (4.4)	-3.08	0.01
HAD dep	4.5 (3.1)	6.0 (3.5)	-3.84	0.001
HAD Total	12.45 (6.1)	15.52 (7.2)	-3.65	0.01
LTE	0.75 (1.0)	0.79 (1.1)	-0.88	0.45
Mastery	20.68 (3.1)	19.43 (3.1)	2.99	0.01
EDE-Q4 Total	2.63 (1.0)	2.21 (1.1)	2.46	0.01

Table 4.1.2 shows the mean and standard deviation of the study variables n = 40. There are significant differences in all variables, as measured by a t-test. There are also highly significant differences between the means when n = 71 and n = 40.

At baseline the sub group increasing in weight have the highest BMI, when compared to the other subgroups, which falls in the overweight category as specified by WHO (1998). Subjects were high in dietary restraint and bingeing behaviour and reach a clinical level of anxiety. Life events were within normal limits for the general population. Lower mastery than other groups and high global eating pathology were

evident. Interestingly despite the level of anxiety cortisol secretion at baseline was the lowest of all groups. During the stress period, this group reacted to the examination stress with the largest increase in cortisol secretion, anxiety and depression, a reduction in dietary restraint and a concomitant reduction in bingeing and global eating behaviour, all of which were highly significant. This was coupled with a significant decrease in mastery. These changes could have significant consequences on health both in the short and long term, for example, arteriosclerosis, hypertension and type II diabetes. There are also implications for performance during the examination although this was not a factor investigated in this study.

4.1.a.1) Characteristics of the decreased weight group (n = 19).

Table 4.1.3: Mean and standard deviation T_1 and T_2 of the main predictors in the study and the dependent variable (BMI), for the 19 individuals who decreased weight.

	T_1 Mean (SD) n = 19	T_2 Mean (SD) n = 19	t	Significance (2 tailed) T_1 vs T_2
Height	1.64 (0.07)			
AUC	89.50 (26.9)	95.56 (35.4)	-1.01	0.33
Restraint	1.31 (1.1)	1.57 (1.4)	-2.53	0.05
BMI	24.08 (3.2)	23.63 (3.2)	8.10	0.001
Binge	0.26 (0.6)	0.11 (0.5)	1.14	0.27
GHQ	2.63 (3.6)	3.89 (4.0)	-1.55	0.14
HAD anx	6.74 (4.4)	8.26 (5.0)	-2.48	0.05
HAD dep	3.05 (3.6)	4.47 (4.2)	-2.41	0.05
HAD Total	9.79 (7.9)	12.74 (8.7)	-2.81	0.05
LTE	1.05 (1.3)	0.93 (1.2)	0.73	0.42
Mastery	21.90 (3.5)	22.21 (3.5)	-0.88	0.39
EDE-Q4 Total	1.84 (1.1)	1.89 (1.3)	-0.25	0.80

At baseline (T_1) the sub group that decrease in weight have the mean lowest BMI (24.08), which falls in the ideal weight category (WHO 1998). Cortisol secretion was high at baseline and anxiety was low when compared to the other subgroups. Both bingeing and global eating pathology were evident and higher than the group whose weight remained the same. They were high in mastery and not surprisingly had no significant psychological problems. During the stress period, this group reacted to the examination stress with a small increase in cortisol secretion, which was not significant. An increase in dietary restraint and a concomitant reduction in bingeing behaviour and global eating behaviour were seen. This was consistent with the group losing weight, and was coupled with a significant rise in anxiety, to possible clinical

levels and depression to a sub threshold clinical level. There was also a small increase in mastery, but this was not significant.

4.1.a.2) Characteristics of the group who remained at the same weight.

Table 4.1.4: Mean and standard deviation of the main variables in the study, for the 12 individuals whose weight remained the same.

	T_1 Mean (SD) n = 12	T_2 Mean (SD) n = 12	t	Significance (2 tailed) T_1 vs T_2
Height	1.62 (0.08)			
AUC	82.92 (31.8)	85.05 (30.7)	-0.79	0.45
Restraint	0.91 (1.2)	0.91 (1.2)		
BMI	24.23 (3.8)	24.23 (3.8)		
Binge	0.08 (0.3)	0.17 (0.4)	-0.56	0.59
GHQ	1.92 (2.6)	3.83 (3.6)	-3.07	0.01
HAD anx	7.25 (3.5)	8.25 (4.1)	-1.91	0.08
HAD dep	3.25 (3.3)	4.33 4.1)	-1.29	0.22
HAD Total	10.5 (6.2)	12.58 (7.8)	-2.38	0.04
LTE	0.92 (1.2)	0.98 (1.3)	-0.84	0.43
Mastery	22.17 (2.6)	21.42 (2.3)	1.15	0.28
EDE-Q4 Total	1.56 (0.9)	1.43 (1.0)	0.45	0.66

At baseline the subgroup remaining the same weight have a mean BMI a little above that of the decreased weight group, which falls in the ideal weight category (WHO 1998). Cortisol secretion was lower than in the increased weight group but higher than decreased weight group. They had no significant psychological problems and they were high in mastery. Both bingeing and global eating pathology were lowest in this group, which would be expected due to the stability in weight measurement and BMI remaining unchanged during the stress period. Restraint remained at the same low level and bingeing showed a small insignificant increase. There was a rise in anxiety to

possible clinical levels and depression increased to sub "caseness" levels, which was significant. A small reduction in mastery was insignificant.

4.1.b) Discussion.

4.1.b.1) Comparison of the three subgroups

This discussion will explore any defining attributes within the three sub groups that might enable identification of individuals that are likely to change weight in response to stress. This information would be of value in identifying individuals who may be subjected to health risks as a result of weight change, during periods of increased stress, for example in individuals who attend clinics or classes seeking help with life style changes such as dieting, smoking cessation and alcohol or other addictive substance use.

The main variables in this study were, cortisol secretion, dietary restraint, body mass index (BMI), bingeing behaviour, mental health, life stressors, mastery and coping strategies and global eating behaviour. These were measured at T_1 (baseline), and T_2 (stress period).

BMI at baseline was highest in the group that increased in weight (BMI 26.1), lowest in the group that lost weight (BMI 24.1), and (BMI 24.2) in the group who remained at the same weight. The range between baseline and the stress period was greatest in the group that increased in weight (+ 0.76), and in the group that lost weight (- 0.45).

Levels of cortisol secretion are lowest at baseline in those who increased weight with the range (23.87nmol/l), between baseline, and the stress period. Those who lost weight had the highest levels of cortisol secretion at baseline rising to the highest level of secretion of the three groups during the stress period (range 6.06 nmol/l). The range (2.13 nmol/l) of cortisol secretion was smallest in the group who remained the same weight throughout the study.

Dietary restraint at baseline was reflected in the BMI of the two groups that changed weight. Restraint score in the increased weight group was almost double that of the group who lost weight being 2.43 (0.90) and 1.31 (1.10) respectively. Interestingly, the changes in BMI and restraint were inversely proportional in that restraint decreased as weight increased and increased as weight decreased in the respective groups. When BMI remained unchanged restraint score was also low.

As would be predicted bingeing behaviour was inversely proportional to dietary restraint. As dietary restraint increased in the decreased weight group, bingeing decreased and vice versa with the increased weight group.

Global mental health as measured by the GHQ-12 scores at baseline, indicated all groups to be below a level of psychological "caseness". "Caseness" refers to a level where the individual is suffering from significant mental health problems such as depression. The group that increased in weight scored 4.57 (3.34), during the stress period, which indicates a significant level of psychological "caseness". The group who lost weight and those who remained the same weight both increased their scores on the

GHQ-12 to just below "caseness". This is an interesting observation in it shows the impact of an exam on general well being.

Anxiety and depression were measured using the HADS-A and HADS-D scales respectively. A score of 8 or more is used to classify cases of clinically relevant levels of anxiety and depression. All groups at baseline and during the stress period scored below the cut off point of 8 for depression. Those who increased in weight had possible clinical levels of anxiety at baseline and this increased during the stress period. The groups who lost weight and remained the same weight were below the cut off point at baseline and reached possible levels of clinically relevant anxiety during the stress period.

Mastery as measured by the mastery scale determined all groups to have high levels of mastery at baseline. During the stress period the increased weight group reduced significantly ($p < 0.01$), in mastery, whilst the group that remained the same weight showed a smaller reduction in mastery, which was not significant. Conversely the decreased weight group increased in mastery but this was also not significant.

Global eating behaviour as measured by the EDE-Q4 showed a significant decrease during the stress period in the increased weight group. The other groups showed no significant differences between the two time periods.

4.1.b.2) Conclusion

The individuals displaying the greatest change in response to the stress period were those who increased in weight. This group at baseline can be identified as having a BMI in the overweight category. They are dieters as identified by a high level of dietary restraint and they have a high level of bingeing behaviour. They may have a clinical level of anxiety and a low level of mastery and coping behaviour. There may also be a significantly higher level of eating pathology identified by the global EDE-Q4. In this study we identified restraint and binge eating as variables of interest and measured these using the EDE-Q4. The EDE-Q4 also measures weight, shape and eating concerns, reflected in the global EDE-Q4 score.

4.1.c) Defining attributes at baseline, of those who increased in weight

- A BMI in the overweight category
- A large increase in cortisol secretion
- A high level of dietary restraint
- A high level of bingeing behaviour
- A high level of EDE-Q4 global eating pathology
- A high level of anxiety, which may be at clinical levels
- A low level of mastery and coping behaviour

The increases in cortisol, BMI and psychological determinants measured in these individuals exposes them to significant health risks in the short term. The purpose of the next section is to determine if this risk is short term or long term. This will be

explored by observing the effects of the stressor post examination in the subjects (n = 18), who completed the T_3 measurements during the summer following the exam.

The next section (4.2), will explore the expected return to baseline of the variables measured at T_2. This will establish if changes at T_2 are transient or long term.

Section 4.2.

4.2.a) Introduction: Investigation of a return to baseline in subjects completing T_3.

Section 2, explores whether there is a return to baseline of the 18 subjects in the first cohort, who completed all three-time points T_1, T_2 and T_3. The students sat their examination in May and the T_3 samples were sent in to the University in July at least eight weeks after the exam. It is anticipated that any changes in variables measured, induced during the stress period, will return towards baseline at T_3. Means and standard deviations are shown in Table 4.2.1 below, a correlation table follows to explore potential relationships between the predictor variables and the dependent variable. This will be followed by two t-tests, firstly to establish changes at T_2 from baseline (T_1), and secondly to establish if there is a return back to baseline at T_3.

Table 4.2.1: Mean and standard deviation of the main predictors and the dependent variable (BMI), for the 18 individuals in cohort 1 that completed all three sampling times. (n = 18)

	T_1 Mean (SD) n=18	T_2 Mean (SD) n=18	T_3 Mean (SD) n=18
Height	1.64 (0.1)		
AUC	97.74 (15.8)	122.42 (24.35)	108.70 (14.09)
Restraint	2.06 (1.0)	1.44 (1.0)	1.89 (0.9)
BMI	26.09 (4.7)	26.26 (5.2)	26.15 (4.6)
Binge	0.53 (0.5)	1.17 (2.0)	0.50 (0.8)
GHQ	1.78 (2.4)	4.17 (3.5)	2.27 (1.5)
HAD anx	6.72 (3.6)	8.83 (5.1)	6.77 (2.5)
HAD dep	3.72 (3.2)	5.22 (3.8)	3.83 (3.5)
HAD Total	10.44 (6.3)	14.06 (8.5)	10.61 (6.2)
LTE	1.05 (1.2)	0.95 (1.1)	1.0 (1.2)
Mastery	20.56 (3.3)	20.11 (3.3)	20.49 (3.0)
EDE-Q4 Total	2.52 (1.3)	2.57 (1.3)	2.41 (1.2)

Cortisol secretion is elevated at T_2 indicating that the examination was a significant stressor. The List of Threatening experiences mean score is within the limits identified from previous stress studies, indicating social and environmental pressures are unlikely to cause this rise in cortisol. At T_3, cortisol levels remain raised but are returning towards baseline. Dietary restraint is lower at T_2 returning towards baseline at T_3. This may reflect the pattern of disinhibition of dietary restraint seen in the whole group and the subgroup that increased in body weight. Bingeing behaviour is inversely proportional to the change in dietary restraint and may reflect the increase in BMI at T_2, that returns towards baseline at T_3. Anxiety and depression increase at T_2 and global mental health as measured by the GHQ increases to a level indicative of a potential psychiatric problem. This is transient indicated by the return to base line levels at T_3. Mastery and coping behaviour are reduced at T_2 returning towards baseline levels at T_3.

Changes in the predictor variables would support our hypothesis and therefore model that as stress increases, dietary restraint is inhibited and bingeing behaviour increases. With decreasing levels of mastery and coping, levels of anxiety and depression increase with global mental health problems being significantly increased. These changes in the predictor variables are transient, which is signified by the return towards baseline of these variables at T_3. The following T-tests are presented to establish any support for these assumptions generated from descriptive data.

Table 4.2.2: Bivariate correlations of all study variables at T_1,T_2 and T_3 n = 18

		BMI	AUC cortisol	Dietary Restraint EDE-Q4	Binge Eating EDE-Q4	EDE-Q4 Global	mastery	GHQ
BMI	T_1							
	T_2							
AUC cortisol	T_1	- .05, p = .85						
	T_2	.14, p = .57						
	T_3	-.15, p = .55						
Dietary Restraint EDE-Q4	T_1	.40, p = .10	.06, p = .81					
	T_2	-.17, p = .49	-.62, p < .01**					
	T_3	.44, p = .07	-.42, p = .08					
Binge Eating EDE-Q4	T_1	.57, p < .01*	-.25, p = .32	.35, p = .15				
	T_2	.64, p < .01**	.28, p = .26	-.17, p = .50				
	T_3	.69, p < .001**	-.06, p = .80	.37, p = .13				
EDE-Q4 Global	T_1	.62, p < .01**	-.27, p = .27	.69, p < .01**	.75, p < 0.001**			
	T_2	.40, p = .10	-.22, p = .39	.47, p < .05*	.70, p < 0.001**			
	T_3	.58, p < .001	-.25, p = .31	.68, p < .01**	.75, p < 0.001**			
Mastery	T_1	-.52, p < .05*	-.08, p = .76	-.46, p = .054	-.52, p < .05*	-.52, p < .05*		
	T_2	-.60, p < .01**	-.03, p = .91	.06, p = .80	-.49, p < .05*	-.32, p = .20		
	T_3	-.52, p < .05*	-.05, p = .84	-.38, p = .12	-.57, p < .05*	-.45, p = .06		
GHQ	T_1	.36, p = .15	.05, p = .85	.26, p = .29	.17, p = .50	.53, p < .05*	-.16, p = .52	.
	T_2	.37, p = .14	-.02, p = .94	.24, p = .35	.15, p = .54	.26, p = .30	-.41, p = .10	
	T_3	.26, p = .29	-.10, p = .72	.08, p = .76	.41, p = .10	.28, p = .27	-.43, p = .08	
HAD Total	T_1	.34, p = .16	-.10, p = .70	.50, p < .05*	.27, p = .27	.62, p < .01*	-.46, p = .054	.75, p < .001**
	T_2	.40, p = .10	.04, p = .87	.07, p = .68	.37, p = .13	.48, p < .05*	-.55, p < .05*	.80, p < .001**
	T_3	.45, p = .06	-.27, p = .28	.28, p = .26	.46, p = .054	.51, p = .05*	-.38, p = .11	.50, p < .05*

. Correlation is significant at the 0.05 level (2 tailed).. Correlation is significant at the 0.01 level (2 tailed).*

4.2.b) Summary of correlations

The increase in cortisol secretion is significantly related to the inhibition of dietary restraint at T_2, whereas there is no correlation at T_1 or T_3. The increase in binge eating is positively correlated with BMI at T_2 and T_3, although at T_3 the level of binge eating has returned back to baseline values.

Mastery is negatively correlated with BMI at T_1, increasing to a highly significant negative correlation at T_2. Mastery is also negatively correlated with anxiety at T_2 only.

Anxiety is correlated with dietary restraint at T_1 and with global mental health at all three time points.

The global EDE-Q4 scores for eating behaviour such as eating concern, weight concern and shape concern is correlated with bingeing behaviour and restraint as would be expected.

These correlations will be explored in the following t-tests.

Table 4.2.3: Mean standard deviation and t-test data of the main predictors in the study and the dependent variable (BMI) at T_1 and T_2, for the 18 individuals who completed all three measurement points.

	T_1 Mean (SD) N=18	T_2 Mean (SD) N=18	T_3 Mean (SD) N=18	t T_1 Vs T_2	t T_1 Vs T_3
Height	1.64 (0.1)				
AUC	97.7 (16)	122.4 (24)	108.7(14)	-3.8***	-2.4*
Restraint	2.1 (1.0)	1.4 (1.0)	1.9 (0.9)	2.5*	0.43
BMI	26.1 (4.9)	26.3 (5.2)	26.2 (4.6)	-1.0	-1.7
Binge	0.5 (0.5)	1.2 (2.0)	0.50 (0.8)	-3.3**	0.8
GHQ	1.8 (2.4)	4.2 (3.5)	2.3 (1.5)	-3.2**	-0.92
HAD anx	6.7 (3.6)	8.8 (5.1)	6.8 (2.5)	-2.8*	-0.8
HAD dep	3.7 (3.2)	5.2 (3.8)	3.8 (3.5)	-2.6*	-0.14
HAD total	10.4 (6.3)	14.1 (8.5)	10.6 (6.2)	-2.6*	-0.1
LTE	1.1 (1.2)	1.0 (1.1)	1.0 (1.2)	0.96	-0.95
Mastery	20.6 (3.3)	20.1 (3.3)	20.5 (3.0)	-1.5	0.8
EDE-Q4 total	2.5 (1.3)	2.6 (1.3)	2.4 (1.2)	-0.7	1.2

*** $p < .001$
** $p < .01$
* $p < .05$

A t-test identifies significant increases in cortisol, anxiety, depression and global mental health changes between T_1 and T_2. Dietary restraint is significantly reduced whilst bingeing behaviour reaches a borderline significant increase. BMI, mastery and global eating behaviour do not significantly change between T_1 and T_2.

A t-test shows that cortisol secretion is reduced in significance and potential psychological problems have declined to being non significant between T_2 and T_3. Restraint and bingeing behaviour are now not significantly different from baseline and this may reflect the return towards baseline of BMI.

4.2.c) Summary of section 4:2.

The descriptive statistics above provide some support for the initial assumption made at the outset of the study, namely, that the increase in cortisol secretion indicates that the examination was a significant stressor. At T_3 cortisol levels remain raised but are not as significantly raised as they were at T_2. Dietary restraint is significantly reduced at T_2 with no significant difference between T_1 and T_3. This reflects the pattern of disinhibition in dietary restraint seen in the whole group and the subgroup that increased in body weight. Bingeing behaviour is inversely proportional to the change in dietary restraint and may reflect the increase in BMI at T_2, although BMI does not change significantly at T_2 in this group. Anxiety and depression increase significantly at T_2 as do global mental health changes as measured by the GHQ. This is transient as shown by the return to below baseline levels at T_3. Mastery and coping behaviour were not significantly altered between the three time points.

These statistics reflect a sample of only 18, but it may be that the changes seen in this group in terms of T_3 and a return of variables to baseline levels, can be applied to the whole group. The study suggests there are implications for short term health issues, both psychological and somatic. There are also implications for student performance during unseen examinations and learning support strategies and psychological support initiatives during this time.

Chapter 5

5.1) Discussion of findings from the main study

There were significant differences in all variables measured between baseline and the exam period. Cortisol secretion, BMI, bingeing behaviour, global mental health and anxiety and depression increased significantly between these two time points ($p < 0.001$). Variables, which reduced significantly, were, dietary restraint ($p < 0.001$), mastery ($p < 0.01$) and global eating behaviour ($p < 0.05$). Significance levels were calculated using paired t-tests.

An increase in body weight is strongly associated with an increase in cortisol secretion and bingeing behaviour. These changes occur in the presence of a reduction in dietary restraint. These changes are not observed in those individuals whose body weight remained the same or decreased. These observations form the most important central findings from the main tenets developed in the origin of this study, and will be discussed and compared to reports from existing literature.

More specifically, an increase in cortisol secretion was strongly associated with an increase in bingeing behaviour and a concomitant reduction in dietary restraint. The stress of the examination resulted in increases in the consumption of high carbohydrate and saturated fat foods and alcohol consumption.

A reduction in restraint was strongly associated with an increase in global eating behaviour symptoms such as concerns about body shape and weight, bingeing and amount of food consumed. This was coupled with a significant reduction in mastery and increasing global psychiatric symptoms, particularly anxiety and depression. These findings form an interesting emerging observation within the field of stress research into eating disorder and the effects of academic examinations on mental health, and will be discussed and compared to reports from existing literature.

Fig 3.3.2 (page 140), depicts the model generated from the regression analysis, of predictors, mediators and moderators of change in BMI between baseline and the stress period. Dietary restraint and change in cortisol secretion are confirmed as predictors of the change in BMI seen during the stress period. The change in bingeing behaviour is a complete mediator of the effects of change in cortisol secretion on BMI change, and a partial mediator of the effects of dietary restraint at baseline on the change in BMI. Change in food choices, for example to high carbohydrate and saturated fat, is a partial mediator of the effects of cortisol change on change in BMI. Restraint at baseline and the change in cortisol secretion are significant moderators of the change in restraint during the stress period and therefore the change in BMI.

The discussion will present a summary of the results and links to existing literature, followed by implications for theory, practice and future research.

5.1.a) Summary of results and links to existing literature

In the whole study group (n = 71), there was an average increase in body weight of approx 2.5 lbs. In those subjects who increased in weight there was an average increase of 5.5 lbs. In those subjects who decreased in weight there was an average decrease in body weight of around 2.5 lbs. There was no change in BMI in 12 of the subjects.

The observed increase in BMI is most likely associated with the increase in an energy dense diet, which is particularly conducive to a positive energy balance and weight gain. Dietary fat usually contributes more to energy density than carbohydrate, and dietary fat is related to increases in weight (Philip and James 2002; Bray and Popkin 1998). The association of dietary changes in response to stress will be discussed below.

Much of the research, exploring the interaction between stress and eating behaviour is based in a laboratory or clinic setting. Choice of food, such as fat or sugar content (Dallman et al 2003), has often been considered a responsive behaviour to life stress either inadvertently or as a deliberate strategy for coping with stress (Lattimore and Caswell 2004; Folkman & Lazarus 1980). This responsive behaviour could arise from a general effect of stress on food intake, for example through physiological changes. Alternatively, changes in eating behaviour may arise from significant individual differences in responses to stress, such as change in dietary restraint or mood. Stress is widely reported to lead to overeating in some individuals (Yacono, Freeman and Gil 2004), and a consistent finding from studies measuring these individual differences is

that those scoring high in dietary restraint eat more under stress, whereas intake is the same or lower in unrestrained eaters (Lattimore and Caswell 2004).

Salivary cortisol in the whole study group (n = 71) rose by 20% and suggests that the examination was a significant stressor. The rise in salivary cortisol was also a significant predictor of change in BMI. In that there was an increase in cortisol secretion of 34%, in the subgroup increasing in weight, a decrease of 7% in those who lost weight, and an increase of 2% in those who remained at the same weight. These findings support hypothesis 1, that the rise in cortisol is associated with a change in BMI. The greatest rise in cortisol was associated with a rise in BMI in the group that increased in weight and the smallest rise in cortisol was seen in the group whose weight remained the same. Cortisol reactivity i.e. the amount of change in cortisol secretion is implicated by these findings in the change in BMI. These findings build on a number of studies discussed in section 1.3.a and b (Macht et al 2005; Lattimore and Caswell; Dallman et al 2004; Yacono, Freeman and Gil 2004; Wardle 2000). What this study adds to the existing literature is the observation of changes in BMI over time in response to the stressor. Many of the studies sited are cross sectional and imply that BMI may change. For example a consistent finding from studies is that those scoring high in dietary restraint eat more under stress, whereas intake is the same or lower in unrestrained eaters. This study, due to its longitudinal component demonstrates that BMI increases significantly in those individuals with high dietary restraint, in response to the stressor and high cortisol reactivity. This study also demonstrates that the increase in BMI is due to an increased consumption of food, and a change in preference to high CHO and saturated fat foods. The changes in BMI were demonstrated in a subgroup of individuals to be transient.

Within the whole group, the linear regression analysis has confirmed that the increase in cortisol secretion is a significant predictor of change in BMI, and that the change in bingeing behaviour is a complete mediator of the change in BMI. The finding of increased cortisol secretion in response to an examination is consistent with the findings of previous studies using an acute stress viva examination (Koh 2004). In a laboratory study, salivary cortisol was sampled in women exposed to psychological challenges such as deliverance of a video taped speech (Epel 2001). A significant rise was seen during the stress period compared to a rest day.

In the whole study group, there was a highly significant increase in bingeing behaviour. In the group that increased in BMI, bingeing behaviour increased by 201% whilst in those who decreased or stayed the same weight changes in bingeing behaviour was not significant.

Linear regression analysis confirmed change in cortisol and dietary restraint at baseline to be predictors of change in bingeing behaviour. Bingeing behaviour is also a complete mediator of the effects of cortisol change on change in BMI and a partial mediator of the effects of dietary restraint on change in BMI. A model depicting the effects of a stressor on these predictor variables is shown in Appendix 9.

Dietary restraint at baseline and change in cortisol are predictors of change in both BMI and bingeing behaviour. A significant finding in the present study is that change in dietary restraint is also a significant mediator of change in BMI and associated with

a large highly significant negative correlation, and change in bingeing behaviour is a complete mediator of the effects of change in cortisol and a partial mediator of the effects of restraint on change in BMI. These findings support hypothesis 5 "Body Mass Index will be correlated with dietary restraint and bingeing behaviour scores". The regression analysis provided a mechanism to explore further these correlations and develop our model supporting the mediator effects described above and shown in fig 3.3.2 on page140. These findings add to the longitudinal diary study by Yacono Freeman and Gill (2004), in which they found as dietary restraint decreased bingeing behaviour increased over a period of one month.

The relationship between life events and psychopathology has been the subject of extensive research. A large body of evidence supports the association between exposure to adverse life events and the onset of depression (Rijsdijk 2001; Brown 1998; Brown and Harris 1989). The extent to which life events constitute a "cause" of depression rather than a shared vulnerability is controversial (Kessler, 1997). In the present study, The List of Threatening Experiences indicates no significant difference between baseline and the stress period [t -.865, p = .40], with findings consistent with other studies (Michalak et al 2004)

A significant reduction in dietary restraint was seen in the whole group. In the subgroup that increased in weight dietary restraint decreased by 50% which was highly significant. Contrary to our hypothesis, in 12 subjects BMI, remained unchanged, dietary restraint also remained unchanged at 0.91 (s.d. 1.2), which was the lowest dietary restraint group. Dietary restraint at baseline was confirmed as predictor

of change in BMI. Change in dietary restraint during the exam period mediates the effects of dietary restraint at baseline, and change in cortisol secretion (i.e. predictors), during the stress period on change in BMI. The change in cortisol and level of restraint at baseline are also significant moderators of the change in dietary restraint seen during the stress period (see fig 3.3.2 page 140). Therefore with these moderators present, significant changes in BMI would be expected.

Previous studies have reported inconsistent results between restrained eating and cortisol secretion (Anderson 2002). Restrained eating behaviour is thought to be a stressor in itself resulting in high cortisol secretion and may play a role in the development of increased weight and obesity (Heatherton et al 1998). Pirke et al (1990), found no correlation between restrained eating (as defined by the Three Factor Eating Questionnaire-Cognitive Restraint scale (TFEQ-R)), (Stunkard and Messick, 1985), and plasma cortisol, while McLean, Barr and Prior (2001), found restrained eating (as defined by the TFEQ-R), was positively correlated with elevated levels of cortisol. Anderson et al (2002), using the TFEQ-R and the Restraint Scale (RS; Herman and Polivy 1980), found that both measures of restraint were positively associated with elevated levels of salivary cortisol. In their laboratory study, Anderson et al (2002), used a single time point for their measurements, which would not allow them to observe changes over time or changing levels of stress. In the present study, (a longitudinal naturalistic design) utilising the EDE-Q4, we found no significant correlation between salivary cortisol and dietary restraint at baseline or during the stress period. However, important findings from the study were changes in these predictor variables, i.e. change in cortisol secretion and change in restraint, were highly negatively correlated. Indicating a rise in cortisol coincided with a reduction in

dietary restraint score in the whole study group. Furthermore, these changes produced significant alterations in BMI. The utilisation of a naturalistic longitudinal design was a key factor in exploring these relationships and is a significant strength of this study.

Global eating behaviour as measured by the EDE-Q4, decreased significantly, over time. This finding may arise from an amalgamation of dietary inhibition and the observed increase in bingeing seen.

A main effect of time was seen in the analysis of food choice as measured by the Food Factor Questionnaire (FFQ), in a subgroup of subjects (n = 38). Total food intake increased between baseline and the stress period and was highly significant. During the stress period there was a highly significant increase in the consumption of CHO and saturated fat. Linear regression confirmed change in cortisol to be a predictor of change in food choice. Change in food choice during the stress period is also a partial mediator of the effects of cortisol change on change in BMI. Thus an increase in cortisol secretion modulates food choices towards a higher calorific intake, particularly in carbohydrates, saturated fat and alcohol. This finding substantiates hypothesis 2 "Subjects who increase in bingeing behaviour will increase consumption of high carbohydrate and saturated fat foods". The change to more energy dense foods impacts significantly in an increase in BMI in susceptible subjects. The total calorific intake may be an under estimate of the true value at baseline: nonetheless, there is a significant increase during the stress period. It may be of value to incorporate a food diary in future studies to corroborate the findings of the FFQ. The finding of increased calorific intake in response to a stressor is consistent with previous studies (Epel et al 2001; Wardle et al 2000; Oliver and Wardle 1998). Epel, (2001) found that stress-

induced cortisol reactivity was related to greater caloric intake after exposure to a novel laboratory stressor. The "reactivity" Epel alludes to is a median split of the salivary cortisol results, into "high reactivity" and "low reactivity". Subjects with high reactivity consumed more calories than subjects with low reactivity. Macronutrient intake was also monitored, with high reactors consuming sweeter, saturated fat foods on a stress day, and the converse being evident in low reactors.

It is possible that women more vulnerable to stress, in their mood responses and cortisol reactivity, may be particularly at risk of stress-induced eating and weight gain. In a cross sectional analysis with a longitudinal element, Wardle et al (2000), compared high work stress periods with low work stress periods and found that high workload periods were associated with higher intake as expenditure, saturated fat and sugar intake. There was a significant moderating effect of restrained eating, with a hyperphagic response to work stress in restrained eaters, compared with no effect in unrestrained eaters. Using a brief questionnaire, Oliver and Wardle (1998), measured self reported effects of stress on eating behaviour and food choice. In the majority of respondents, 154 of 212 students, snacking behaviour was reportedly increased by stress. Energy dense foods such as sweets, chocolate, cakes, biscuits and savoury snacks, were reported to be eaten during times of stress by the respondents. The concomitant intake of foods such as meat, fish and vegetables decreased during stressful periods. The direction of change in intake was predicted in part by dieting status, with dieters being more likely to report stress hyperphagia, and the converse being found for non-dieters.

The results from this study strongly suggest that psychophysiological response to stress influences subsequent eating behaviour.

A main effect of time was seen in the analysis of mastery as measured by the Mastery Scale, in the whole study group (n = 71). Mastery decreased from 21.3 (s.d. 3.1), at baseline to 20.5 (s.d.3.3) during the stress period and this decrease was significant [t = 2.61; $p < .01$]. In subjects increasing in BMI, mastery decreased from 20.7 (s.d.3.1) at baseline to 19.42 (s.d. 3.1), during the stress period. This decrease of 6% was significant [t = 2.99; $p < .01$]. In subjects decreasing in BMI, mastery increased from 21.9 (s.d.3.5), at baseline to 22.2 (s.d.3.5) during the stress period and was not significant [t = -0.88; $p = .39$]. In 12 subjects whose weight remained the same, mastery decreased from 22.2 (s.d. 2.6), at baseline to 21.4 (s.d. 2.3). The decrease of 4% was not significant [t = 1.15; $p = .28$]. Mastery was also highly negatively correlated with BMI [Pearson correlation -.40, $p < .01$] during the stress period.

Mastery was not correlated with bingeing behaviour at baseline [Pearson correlation -- .15, $p = .22$], however, during the stress period, mastery is significantly negatively correlated with bingeing behaviour [Pearson correlation -.29, $p < .05$]. This finding supports hypothesis 3, "Subjects who are low in mastery will increase in bingeing behaviour".

The finding of changes in mastery, and therefore in ones ability of global control, are consistent with previous studies (McKean Skaff et al 1996; Cohen and Edwards 1989). Subjects increasing in mastery when conditions change, sustain a consistent sense of control, coupled with low dietary restraint. This may be the focus for low reactivity to the stressor, resistance to bingeing and for a reduction in BMI. Conversely, the

reduction in mastery in the subjects increasing in BMI, who are high in dietary restraint, may be the focus of high reactivity to the stressor, susceptibility to bingeing with the concomitant increase in BMI. Mastery was also highly negatively correlated with global mental health [Pearson correlation -.51, $p < .001$], further compounding the effects driving an increase in BMI, just described; this will be discussed further in the next section.

Global mental health was measured using the General Health Questionnaire – 12, and was seen to increase from baseline to the stress period in the whole group. This increase was highly significant at. The regression analysis did not confirm the change in GHQ as a mediator or moderator of the effects of stress on change in BMI. However a reading of 4.0 and above is considered to be "caseness". Therefore, it is possible that some of the subjects were suffering from a significant increase in mental health problems.

Anxiety and depression were measured using the Hospital Anxiety and Depression Scale (HADS). A score of 8 or more is used to classify possible cases of clinically relevant levels of anxiety and depression. There was a highly significant increase in anxiety between baseline and the stress period. Depression also showed a highly significant increase between baseline and the stress period. Thus, subjects rated anxiety ("caseness"), just below the cut off point at baseline, and above during the stress period. There may be clinical levels of anxiety in some subjects at both time points, the change however is highly significant and indicates a significant increase in anxiety symptoms. Regression analysis did not confirm the change in HADS, as a mediator or moderator of the effects of stress on the change in BMI.

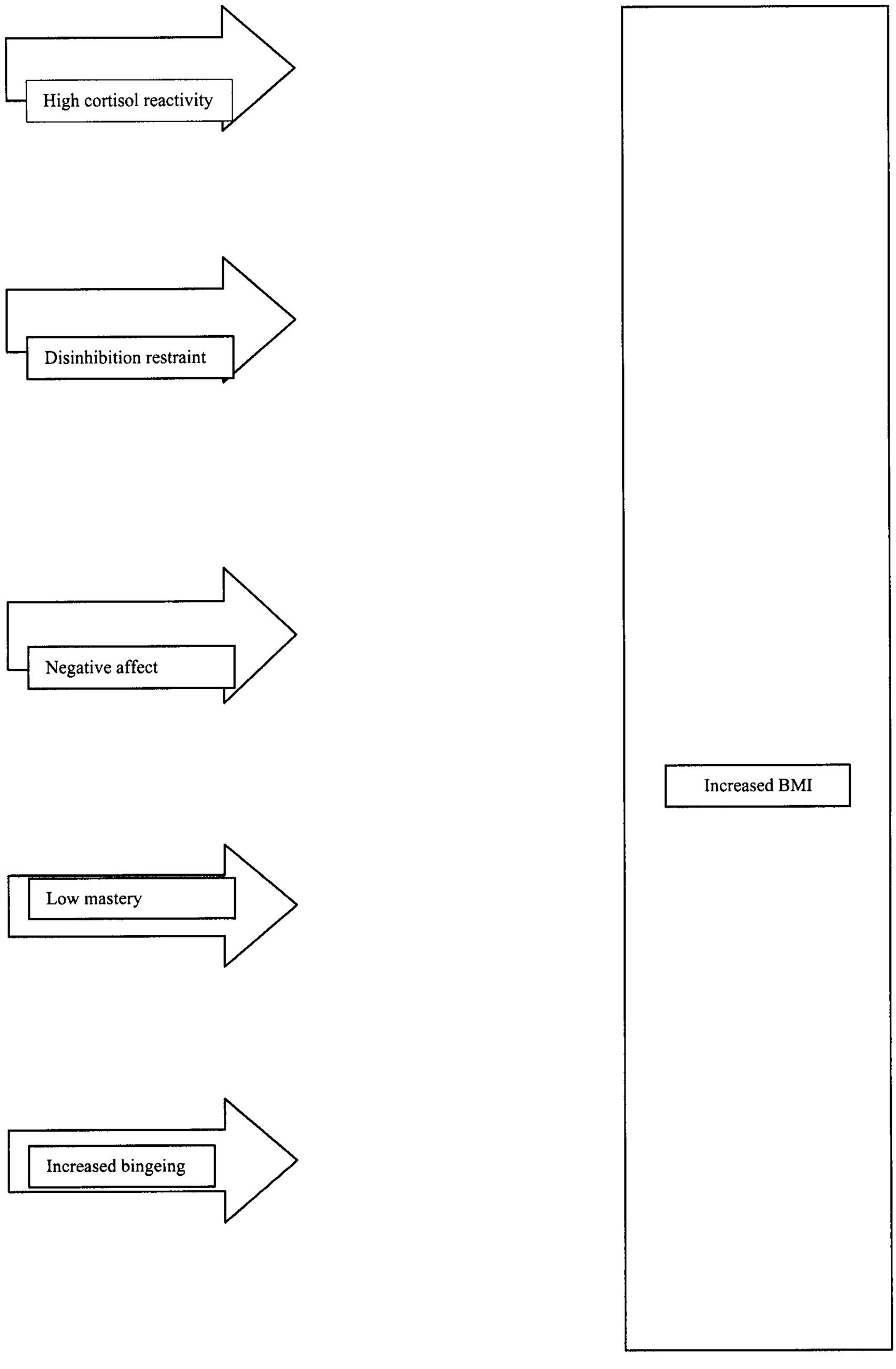
The finding that negative affect is correlated with dietary restraint and bingeing behaviour, is consistent with previous studies. Distress has been found to suppress eating in unrestrained eaters, and increases eating in restrained eaters (Baucom and Aiken 1981, Heatherton, Herman, and Polivy 1991, Heatherton, Polivy, Herman and Baumeister 1993, Polivy and Herman 1999). Restrained eaters have also been found to demonstrate counterregulatory eating behaviours when anxious or stressed (Baucom and Aiken, 1981; Polivy and Herman, 1999; Tanofsky-Kraff et al, 2000; Shapiro, and Anderson, 2004). In many of these studies, the laboratory "taste-test" paradigm has been utilised. Data obtained using this methodology have identified that restrained eaters paradoxically consume more calories than non-restrained eaters when exposed to emotional stress. In a longitudinal study Stice et al's (1998), findings converge with those from laboratory studies in suggesting that negative affect moderates the relation between dieting and binge eating. In a field study, students three to four days before an academic examination showed higher ratings of tension, fear and emotional stress as well as lower ratings of happiness, relaxation and positive mood, on a self rating scale (Macht, et al 2005). These students also reported a higher tendency to eat in order to distract themselves from stress. It is suggested that eating may distract from the experience of negative emotions (Spitzer and Rodin 1983; Macht, et al 2005). The present study found no evidence of moderation but did find significant correlations between affect, restraint and bingeing behaviour, in their effects on the dependent variable change in BMI. The results of these studies, coupled with the findings from the present study, support the contention that dieting is related to binge eating and that negative mood precipitates disinhibited eating among restrained eaters, but not non-restrained eaters. An interesting additional finding in the present study was that there is an increase in dietary restraint in subjects who decreased in BMI.

In addition to these findings there is literature which suggests that some individuals increase their caloric intake during times of stress while others reduce their intake. The "comfort food" hypothesis supports the notion that a high carbohydrate and saturated fat diet is consumed during times of stress to ameliorate the psychophysiological effects of the stressor. This could be as part of avoidance behaviour in order to reduce the impact of stress, for example "comfort food" may enhance mood, at least in the short term (Macht and Simons 2000). Mastery and coping skills may also impact on cognitive decision making when choosing food type and the amount consumed; individuals with low mastery and coping skills may be more likely to indulge in "comfort eating".

Data from the present study are generally supported by previous studies (discussed above), using both laboratory and naturalistic designs. However, the findings from the group who increase in BMI (n = 40), show even stronger evidence for the relationship between negative affect, mastery, restraint and binge eating and an increase in the dependent variable BMI. There was a highly significant increase in the HADS scores for anxiety and depression in this group (n = 40), during the stress period. Global mental health as measured by the GHQ-12 also showed a highly significant increase during the stress period, indicating significant psychological problems might be present.

The findings from the present study are presented in Fig 5.1.1 below which depicts changes in predictor variables in response to a stressor culminating in an increase in BMI.

Fig 5.1:1 Predictor variables which have significantly correlated with an increase in BMI.



5.2) Implications of bingeing behaviour, weight increase and obesity.

This study has shown that the predictor variables discussed in the previous section and presented in fig 5.1.1 are significant antecedents associated with the occurrence of an increase in BMI. The logical conclusion from this is that individuals who are high cortisol reactors, and high in dietary restraint are especially vulnerable to adverse effects of stress on health, through influences on the quantity and quality of food intake. These results suggest a psychophysiological response to stress influencing subsequent eating behaviour. This is considered to be one of a range of health related behaviours that might be responsive to life stress or emotional well being, either inadvertently or as a deliberate strategy in coping with stress (Folkman and Lazarus 1980). Over time, these responses to stress in vulnerable individuals particularly at risk of stress induced eating and weight gain, could impact on general health. High cortisol reactors are also at risk of developing abdominal obesity, through a direct action of cortisol on stored energy (Dallman et al 2004). Abdominal obesity is associated with greater health risks than that in peripheral regions and is an independent risk factor for the development of risk factors and morbidity, even when BMI is not markedly increased (National Heart, Lung and Blood Institute, 1998). There is also an increased propensity to develop peripheral vascular disease and type II diabetes.

5.3) Limitations of the study.

There are some weaknesses in the present study.

The target population for this study was students attending a BSc (hons) course at a London University. From a potential 225 subjects 84 consented to undertake the study and 13 ultimately dropped out. There is no information available from the 141 who

declined consent for the study. It would be interesting to follow up those subjects who declined consent to establish, why they did not consent and if possible obtain some baseline measures. For example, on talking to the subjects when returning questionnaires and saliva samples, the most common comment was a dislike of the "feeling" of cotton wool in their mouth. This may have been one reason for refusing to participate. In future studies, a follow up attempt could be aimed at completing questionnaires only. This would provide information on all variables except salivary cortisol.

Limitations to the number of variables included were determined in part, by the statistical design of the study. The inclusion of additional independent variables in the regression equation is likely to yield a less valid model of prediction. The sample size would have to be increased considerably to allow the inclusion of a larger number of independent variables. Increasing the number of assessments also puts additional demands on the participating subjects.

The numbers in the subgroups are low, particularly in the group that reduced in weight (n = 19), and remained the same weight (n = 12). However, the fact that significant differences were found in the face of low power suggests that the predictors have a significant effect on change in body weight, particularly an increase in body weight where n = 40.

The EDE-Q4, global measurement shows a reduction in global pathology at T_2, despite bingeing behaviour increasing and dietary restraint reducing. This suggests that the global measurement is not a good measure of eating pathology.

The food factor questionnaire was administered to 38 subjects. Administration to all subjects would have allowed further power in the regression analysis on the role of FFQ as a mediator of the effects of stress on change in body weight.

The use of self report questionnaires is often questioned in terms of reliability and validity. They do however provide a useful alternative to interviews and subjects are more likely to complete a questionnaire, rather than attend an interview. The Cronbach Alpha scores in the present study suggest the questions were answered with a good degree of consistency. There are also reliability and validity studies to support the use of these questionnaires.

Volunteers may be representative of those who cope well with adversity and are therefore more likely to volunteer for a study they think may help researchers establish how stress is related to health. Additionally, although the principal researcher was not a unit leader in the subject's academic course, there was some involvement in delivering specialist life science topics. Whilst this was not a considerable proportion of the units, the students may have felt that they would like to support the general idea of assisting with a research project.

Although mastery appears to reflect an important difference between groups it is not clear how this might impact on weight change. Women who binge may do so because they believe the situation to be uncontrollable or unsolvable, or because they perceive themselves to be ineffective (Lazarus and Folkman, 1984; Bandura, 1977). Identifying which of these is correct would inform treatment more than simply saying, "there

appear to be differences in mastery and coping between those who binge and those who do not". Mastery, depression and anxiety, were seen to be more significantly correlated during the stress period. Perhaps what is needed is to be given confidence in their mastery and coping ability.

5.4) Strengths

This longitudinal naturalistic approach of this study facilitates investigation of the predictive effects of stress, dietary restraint, mastery, mood and bingeing behaviour on body weight. The design facilitates the use of regression analysis to examine moderator and mediator effects of the predictors on the dependent variable. This study builds on EPEL (2001), and Macht (2005), and other studies cited in the relevant subsections above. In many of the cross sectional laboratory and naturalistic studies cited, findings are limited to the inferences that could be drawn from the observed association between predictor variables and the dependent variable.

The use of a longitudinal design helps to address questions relating to direction of causation and the predictive implications of restraint, stress, mastery and food choice on changes in body weight. Data from this study adds to the literature on identification of the defining psychophysiological, attributes in subjects at risk of change in body weight during times of stress.

Another strength of the study is the low drop out rate of 15% in this study is low. This was facilitated by the support of colleagues in maintaining interest and motivation in the completion of the assessment regime.

5.5) Implications: Notwithstanding the limitations described above, theory, research, practice and conclusions.

This study raises important questions related to the individual attributes of subjects whose response to stress is a change in eating behaviour, body weight and mental health.

There are significant implications of hypercortisolaemia, an increase in bingeing behaviour and negative affect, on health. Namely, an increase in central body fat with a tendency towards obesity, hyperlipidaemia increasing the risk of peripheral vascular disease, hyperglycaemia increasing the risk of type II diabetes and an increase in anxiety and depression to clinical levels.

These changes coupled with low mastery and coping skills pose significant risk factors for health. Initially these changes would appear to be transient and in the short term the results would suggest that the effects might be short lived. However, repeated exposure to stress in some individuals may increase the risk of significant enduring pathological changes affecting health.

Therefore the study findings would support the use of brief interventions for individuals susceptible to the changes described. Brief interventions include, giving susceptible individuals information about stress, and how to cope with it by enhancing abilities such as mastery and coping behaviour. Cognitive behavioural therapy is extremely valuable in helping vulnerable individuals to cope with stress. These

interventions have been shown to be effective in reducing psychophysiological responses to stress such as hypertension, alcoholism and depression.

The findings of this study have identified significant predictors of change in BMI. The design of the study has enabled the identification of individual differences, which are moderators, or mediators of the effects of stress on change in BMI (see fig 5.1.1 page 174). Identification of these individual differences in subjects presenting for example at smoking cessation programmes, or alcohol reduction programmes, may improve the likelihood of a successful outcome. Subjects who volunteer for these programmes are more likely to be successful in the outcome if they are not subjected to high cortisol reactivity, changes in dietary restraint, increases in anxiety and depression, poor coping skills, low mastery and episodes of bingeing behaviour leading to significant changes in body weight. Research in this area aimed at identifying vulnerable subjects, predisposed to these changes would enable the provision of individualised care and interventions. This may improve the likelihood of a successful outcome, and enhance quality of life in times of stress.

The findings of this study also suggest that students may benefit from the advice and support of their academic tutor counsellor/ personal tutor. In most academic establishments students are assigned, tutor counsellors/ personal tutors. There is evidence that the majority of students do not seek help from these support staff throughout what may be a three or four year course of study. Therefore, it may be beneficial to heighten the awareness of staff and students to the possible detrimental effects of unseen examinations on health albeit only a transient risk in the short term.

The use of unseen examinations is commonplace in institutions of education at all levels in the U.K. The results of this study suggest that the perceived stress of an unseen examination may impact on academic performance in susceptible individuals. It would therefore seem pertinent to explore this theory further firstly to establish any correlation between level of stress and academic performance, and secondly to explore other mechanisms of academic assessment.

References

Abraham, G.E. (1969). Solid phase radioimmunoassay of oestradiol-17 (beta). J. *Clin. Endocrinol. Metab.*, 29: 866-870.

Ahima, R.S., Prabakaran, D. Mantzoros, C. Qu, D. Lowell, B. Maratos-Flier, E. Flier J, S. (1996) Role of leptin in the neuroendocrine response to fasting, *Nature*, 382, 250-252.

Allen, Y.S., Adrain, T.E., Allen, J. et al (1983). Neuropeptide Y distribution in the rat brain. *Science*, 221:877-879.

Anderson, D.A., Shapiro, J.R., Lundgren, J. D., Spataro, L.E. and Frye, C.A. (2002) Self-reported dietary restraint is associated with elevated levels of salivary cortisol. *Appetite*. 38, 13-17

Andres, R. Elahi, D. Tobin, J. D., Muller, D. C., and Brant, L. (1985) Impact of age on weight goals. *Annals of Internal Measurement*, 103, 1030-1033.

Antelman, S. M., & Szechtman, H. Chin, P. & Fisher, A. E. (1975). Tail pinch-induced eating, gnawing, and licking behaviour in rats: Dependence on the nigrostrialtal dopamine system. *Brain Research*, 99, 319-337.

Antelman, S. M., Eichler, A, J., Black, C. A., & Kocan, D. (1980). Interchangeability of stress and amphetamine in sensitization. *Science*, 207, 329-331.

Barsh, G. S., Farooqi, I.S., and O'rahilly, S. (2000) Genetics of body weight regulation, *Nature*, 404, 644-651.

Baucom DH, Aiken PA.(1981) Effect of depressed mood in eating among obese and nonobese dieting and nondieting persons. *J Pers Soc Psychol.* Sep;41(3):577-85.

Bell, M., Bhatnagar, S., Liang, J., Soriano, L., Nagy, T., Dallman, M., (2000) Voluntary sucrose ingestion, like corticosterone replacement, prevents the metabolic deficits of adrenalectomy. *Journal of neuroendocrinology*, May;12(5):461-70.

Bellisle, F. Louis-Sylvestre, J. Linet, N. Rocaboy, B. Dalle, B. Cheneau, F. L'Hinovet, D. and Guyot, L. (1990). Anxiety and food intake in men. *Psychosomatic Medicine*, 52, 452-7.

Berglund, M.M., Hopskind, P.A. and Gehlert, D.R. (2003). Recent developments in our understanding of the physiological role of PP-fold peptide receptor subtypes. *Experimental Biology and Medicine.*, 228:217-44.

Billington, C.J., Briggs, J.E., Grace, M. and Levine, A.S. (1991). Effects of intracerebroventricular injection of neuropeptide Y on energy metabolism. *American Journal of Physiology*: 260:R321-7.

Blundell, J.E. (2000) *A psychobiological system approach to appetite and weight control*, In, *Eating disorders and obesity: a comprehensive handbook*, (2nd ed), Eds Fairburn, C.G. and Brownell, K,D. (2000), The Guilford Press, New York, 43-49.

Blundell, J.E. and Cooling, J. (2000) Routes to obesity: Phenotypes, food choices and activity. *British Journal of Nutrition*, 83 (suppl.1), S33-S38.

Borkovec T, Robinson E, Pruzinsky T, DePree J. (1983) Preliminary exploration of worry: some characteristics and processes. *Behav Res Ther.* 21(1):9-16.

Bouchard, C. et al (1990) The response to long term overfeeding in identical twins. *N. Eng.J.Med,* 322, 1477-1482.

Bouchard, C (2002) *Genetic influences on body weight*, in. *Eating Disorders and obesity, A comprehensive handbook* 2nd Ed, Eds Fairburn, C, G, and Brownell, K, D. The Guildford Press. New York. 16-21.

Boyar, R. M., Hellman, L. D., Roffwarg, H., *et al* (1977) Cortisol secretion and metabolism in anorexia nervosa. N.Engl.J.Med., 296, 190-193.

Bray, G., (1985) Autonomic and endocrine factors in the regulation of food intake. *Brain Research Bulletin* 14 505-510.

Bray, G. A., and Popkin, B.M. (1998) Dietary fat intake does affect obesity rate. *American Journal of Clinical Nutrition*. 68, 1157-1173.

Brown, E. Sherwood; Woolston, Dixie J; Frol, Alan; Bobadilla, Leonardo; Khan, David A; Hanczyc, Margaret; Rush, A. John; Fleckenstein, James; Babcock, Evelyn;

Bruch, H. (1961). Transformation of oral impulses in eating disorders: A conceptual approach, *Psychiatric Quarterly*, 35, 458-481.

Brown, G.W. and Harris, T.O. (1989) *Life events and illness*. Guildford Press. New York

Brown, G.W. (1998) Genetic and population perspectives on life events and depression. *Social Psychiatry and Psychiatric Epidemiology*. 33, 363-372.

Brugha, T., Bebbington, P., Tennant, C. and Hurry, J. (1985) The list of threatening experiences: a subset of 12 life event categories with considerable long term contextual threat. *Psychological Medicine*, 15, 189-194.

Buffentstein, R. Karklin, A. and Driver, H.S. (2000) Beneficial physiological and performance responses to a month of restricted energy intake in healthy overweight women. *Physiology and Behaviour*. 68, 439-444.

Bulik, C. M. Sullivan, P. F. and Kendler, K. S. (2003) Genetic and environmental contributions to obesity and binge eating. *International Journal of Eating Disorders*. 33, 293-298.

Cade, J. E. and Margetts, B. M. (1988) Nutrient sources in the English diet: Quantitative data from three English towns. *International Journal of Epidemiology*, 17, 844-848.

Cannon, W., (1914) The emergency function of the adrenal medulla in pain and in the major emotion. *American Journal of Physiology*. 33. 356.

Cannon, W. B. (1915) *Bodily changes in pain, fear and rage* (2nd ed.). New York: Appleton.

Cannon, W., Paz, D. (1911) Emotional stimulation of the adrenal gland secretion. *American Journal of Physiology*.28. 64.

Carlson, A.J. (1916). *The control of hunger in health and disease.* Chicago: University of Chicago Press.

Cattanach, L., Rodin, J. (1988) Psychosocial components of the stress process in bulimia. *International Journal of Eating Disorders*, 7. 1. 75-88.

Chagnon, Y.C. and Bouchard, C. (1996) Genetics of obesity: advances from rodent studies. *Trends in Genetics*, 12, 441-444.

Cherrington, A. D. (1999) Control of glucose uptake and release in the liver in vivo. *Diabetes*, 48, 1198-1214.

Chalmer, D., Lovenberg, T., De Souza, E. (1995) Localization of novel corticotropin-releasing factor receptor (CRF2) mRNA expression to specific subcortical nuclei in rat brain: comparison with CRF1 receptor mRNA expression. *J Neurosci.* 15(10):6340-50.

Cohen, S. and Edwards, J. R. (1989) Personality characteristics as moderators of the relationship between stress and disorder. In R. W. J. Neufield (Ed), *Advances in the investigation of psychological stress*. New York, Wiley. 235-283.

Compaan, J.C., Groenink, L. van der Gugten, J. Maes, R. A. and Olivier, B. (1996) 5-HT1A receptor agonist flesinoxan enhances Fos immunoreactivity in rat central amygdala, bed nucleus of the stria terminalis and hypothalamus. *European Journal of Neuroscience*, 11, 2340-7.

Comuzzie, A. G., and Allison, D. B. (1998) The search for human obesity genes. *Science*, 280. 1374-1377.

Connan, F. Campbell, I.C., Katzman, M. Lightman, S.L., and Treasure, J. (2003) A neurodevelopmental model for anorexia nervosa. *Physiology and Behaviour*, 79, 13-24.

Corney, R.H. & Clare, A.W. (1985) The construction, development and testing of a self-report questionnaire to identify social problems. *Psychological Medicine*, 15: 637-649.

Coyne, J, and Downey, G. (1991) Social factors and psychopathology: Stress, social support and coping processes. *Annual Review of Psychology*, 42, 401-425.

Cullum, C. Munro. (2004) Hippocampal volume, spectroscopy, cognition, and mood in patients receiving corticosteroid therapy. *Biological Psychiatry*, 55,5, 538-545.

Dallman, M. F., La Fleur, S. E., Pecoraro, N. C., Gomez, F. Houshyar, H. Akan, S. F. (2004) Glucocorticoids: food intake, abdominal obesity and wealthy nations in 2004. *Endocrinology mini-review.*

Dallman, M. F., Akana, S. F., Laugero, K. D., Gomez, F. Manalo, S. Bell, M.E. and Bhatnagar, S. (2003) A spoonful of sugar: feedback signals of energy stores and corticosterone regulate responses to chronic stress. *Physiology and Behaviour*, 79, 3-12.

Dess, N. K., Choe, S. & Minor, T, R. (1998) The interaction of diet and stress in rats; high energy food and sucrose treatment. *Journal of Experimental Psychology Animal Behav Process*, 24, 60-71.

DiMaggio, D.A., Chronwall, B.M., Buchman, K. and O'Donohue, T.L. (1985). Pancreatic polypeptide immunoreactivity in rat brain is actually neuropeptide Y. *Neuroscience.*, 15: 1149-1157.

Doerr, P., Fichter, M., Pirke, K. M., *et al* (1979) Relationship between weight gain and hypothalamic pituitary adrenal function in patients with anorexia nervosa. *Journal of Steroid Biochemistry*, 13, 529-537.

Donohoe T. (1984) Stress-induced anorexia: implications for anorexia nervosa. *Life Sci.* Jan 16;34(3):203-18.

Dunn A, Welch J.(1991) Stress- and endotoxin-induced increases in brain tryptophan and serotonin metabolism depend on sympathetic nervous system activity. *J Neurochem.* 57(5):1615-22.

Ely, D., Dapper, V., Marasca, J., Correa, J., Gamaro, G., Xavier, M., Michalowski, M., Catelli, D., Rosat, R., Ferreira, M. and Dalmaz, C. (1997) Effect of restraint stress on feeding behaviour of rats. *Physiol of Behav*, 61, 395-8

Epel, E. Lapidus, R. McEwen, B. Brownell, K. (2001) Stress may add bite to appetite in women: a laboratory study of stress-induced cortisol and eating behaviour. Psychoneuroendocrinology, 26, 37-49.

Fairburn, C. G., and Brownell, K. D. (2002) Eating disorders and Obesity: a comprehensive handbook, 2nd Ed, London. The Guilford Press.

Fairburn, C. G., and Beglin, S.J. (1994) The assessment of eating disorders: interview or self report questionnaire? *The International Journal of Eating Disorders*. 16: 363-370.

Falconer, D. S., and Mackay, T.F.C. (1995) *Introduction to Quantitative Genetics*. Addison-Wesley, Harlow.

Fitzgibbon, M. L., Stolley, M.R., and Kirschenbaum, D.M. (1993) Obese people who seek treatment have different characteristics than those who do not seek treatment. *Health Psychology*, 12, 346-53.

Flatt, J.P., (1989) Effects of corticosterone on RG, food intake, and energy balance. *International Journal of Obesity* 13, 552

Folkman, S., and Lazarus,R. (1980) An analysis of coping in a middle aged community sample. *J Health Soc Behav*, 21, 219-39.

Francis, D. D. and Meaney, M.J. (1999) Maternal Care and the development of stress responses. *Current Opinion in Neurobiology*, 9, 128-134.

Friedman, J. M. and Hallas, J, L. (1998) Leptin and the regulation of body weight in mammals. *Nature*, 395, 763-770.

Friedman, J. M. (2000) Obesity in the new millennium. *Nature*, 404, 632-634.

Friedman, J. M. (2003) A war on obesity, Not the obese. *Science*,299, 856-858.

Garner, D., and Garfinkel, P., (1997) *Handbook of Treatment for Eating Disorders*. New York. Guilford Press.

Geliebter, A. and Aversa, A. (2003) Emotional Eating in overweight, normal weight, and underweight individuals. *Eating Behaviours*. 3, 341-347.

Gelhert, D.R. (1999). Role of hypothalamic neuropeptide Y in feeding and obesity. *Neuropeptides*. 33:329-338.

Gerner, R. H. and Gwirtsman, H. E. (1981) Abnormalities of dexamethasone suppression test and urinary MHPG in anorexia nervosa. Am.J.Psychiatry, 138, 650-653.

Goldberg, D. (1972) *The detection of psychiatric illness by questionnaire* (Maudsley Monograph No 21). Oxford, England. Oxford University Press.

Goldsmith, S.J., Anger-Friedfeld, K. Beren, S. Rudolph, D. Boeck, M. and Aronne, L. (1992) Psychiatric illness in patients presenting for obesity treatment. *International Journal of Eating Disorders*, 12; 63-71.

Greenberg B, Harvey P.(1986) The prediction of binge eating over time. *Addict Behav.* 11(4):383-8.

Greeno, C.G., & Wing, R.R. (1994). Stress-induced eating. *Psychological Bulletin.* 115(3), 444-464.

Grilo, C., Masheb, R., and Wilson, G. (2001) A comparison of different methods for assessing the features of eating disorders in patients with binge eating disorders, *Journal of Consulting and Clinical Psychology*, (69), 317-322.

Grunberg, N. E., & Straub, R. O. (1992). The role of gender and taste class in the effects of stress on eating. *Health Psychology*, 11, 97-100.

Gurin, P. and Brim, O. G. J. (1984) Change in self in adulthood: The example of sense of control. In P. B. Baltes. and O. G. J. Brim (Eds), *Life span development and behaviour*, New York: Academic Press. 281-334.

Gwirtsman, H. E., Kaye, W. H., George, D. T., *et al* (1989) Central and peripheral ACTH and cortisol levels in anorexia nervosa and bulimia. Arch.Gen.Psychiatry, 46, 61-69.

Hahn, T.M., Breininger, J.F., Baskin, D.G. and Schwartz, M.W. (1999). Coexpression of Agrp and NPY in fasting activated hypothalamic neurons. *Nat Neuroscience*: 1:271-2.

Halmi KA.(1996) The psychobiology of eating behavior in anorexia nervosa. *Psychiatry Res*. 62, (1),23-9

Hardy, E., Shapiro, D., Haynes, C., Rick, J. (1999) Validation of the General Health Questionnaire-12 using a sample of employees from Englands Health Care Services. *Psychological Assessment*. 11, 2, 159-165.

Hawkins, R., Clement, P. (1980) Development and construct validation of a self report measure of binge eating tendencies. *Addictive Behaviours*. 5. 219-226.

Hazelwood, R.L. (1993). The pancreatic polypeptide (PP-fold) family: gastrointestinal, vascular, and feeding behavioural implications. *Proc Soc Exp Biol Med*., 202:44-63.

Heatherton, T.F., Herman, C.P., Polivy, J., King, G.A., and McGree, S.T. (1988) The (mis) measurement of restraint: An analysis of conceptual and psychometric issues. *Journal of Abnormal Psychology*, 97, 19-28.

Heatherton, T.F., Herman, C.P. and Polivy, J. (1991). Effects of physical threat and ego threat on eating behavior. *Journal of Personality and Social Psychology*, 60, 138 - 143.

Heatherton, T. F., & Baumeister, R. F. (1991). Binge-eating as escape from self awareness. *Psychological Bulletin*, 110, 86-108.

Heatherton, T.F., Herman, C.P., Polivy, J., King, G.A. and McGree, S.T. (1988) The (Mis)measurement of restraint: an analysis of conceptual and psychometric issues. *Journal of Abnormal Psychology*. 97, 19-28.

Heatherton, T.F. and Polivy, J. (1992). *Chronic dieting and eating disorders: A spiral model.* In: Crowther, J.H., Tennenbaum, D.L., Hobfoll and Stephens, M.A.P. (Eds.), Washington. *The Etiology of Bulimia Nervosa*. Hemisphere Publishing Corporation.

Heatherton T.F. Polivy J, Herman CP, Baumeister RF. (1993) Self-awareness, task failure, and disinhibition: how attentional focus affects eating. *J Pers*. 61, (1), 49-61.

Heinrichs, S.C., Menzaghi, F. Pich, E.M., Haugher, R. L. Koob, G.F. (1993) Corticotropin releasing factor in the paraventricular nucleus modulates feeding induced by neuropeptide Y. *Brain Research*, 611, (1), 18-24.

Herman, C. (1997) International experiences with the hospital anxiety and depression scale- A review of validation data and clinical results. *Journal of Psychosomatic Research*. 42, 1. 17-41

Herman, C, and Mack, D. (1975) Restrained and unrestrained eating. *Journal of Personality*, 43, 647-660.

Herman, C.P., & Polivy, J. (1975). Anxiety, restraint, and eating behaviour. *J. of Abnormal Psychology*. 84(6), 66-72.

Herman, C., Polivy, J., (1980) Stress induced eating and eating induced stress (reduction?): a response to Robbins and Fray. *Appetite*. 1. 135-139.

Herman, C.P. and Polivy, J. (1980) Restrained eating. In A.J. Stunkard (Ed), *Obesity*. 208-225. New York: Saunders.

Herman, C.P., & Polivy, J. (1984). A boundary model for the regulation of eating. In A.J. Stunkard and E. Stellar (eds), *Eating and its disorders*, New York: Raven Press, 141-56

Herman CP, Polivy J.(1988) Psychological factors in the control of appetite. *Curr Concepts Nutr*. 16:41-51

Hill, J,O., and Peters, J,C. (1998) Environmental contributions to the obesity epidemic. *Science*, 280, 1371-1374.

Horwitz, A.V., Scheid, Teresa L (1999). *A handbook for the study of mental health: Social contexts, theories, and systems*. New York, Cambridge University Press. 161-175.

Janzen, B., Kelly, I. And Saklofske, D. (1992) Bulimic symptomatology and coping in a nonclinical sample, *Perceptual and Motor Skills*, 75, 395-399.

Junquera, J. Lanzagorta, G. & Russek, M. (1987). Adrenalin-induced anorexia acts on tail pinch feeding in the rat. *Appetite*, 9, 113-118.

Kahn, S. E., Prigeon, R. L., McCulloch, D. K., Boyko, E. J., Bergman, R. N., Schwartz, M. W., Neifing, J. L., et al (1993) Quantification of the relationship between insulin sensitivity and beta-cell function in human subjects. Evidence for a hyperbolic function. *Diabetes*, 42. 1663-1672.

Kaplan, H. I., & Kaplan, H. S. (1957). The psychosomatic concept of obesity. *Journal of Nervous and Mental Disorders,* 125, 181-201.

Kessler, R. C. (1997) The effects of stressful life events on depression. *Annual Review of Psychology*. 48, 191-214.

Keys, A., Brozek, J., Henschel, A., Mickelson, O., & Taylor, H. L. (1950). *The biology of human starvation* (2 vols.). Minneapolis: University of Minnesota Press.

Klein, L., Faraday, M., Grunberg, N. (1996) Gender differences in eating after exposure to a noise stressor. *Annals of Behavioural Medicine*. 18. S103.

Klem. M. L., Wing, R.R., McGuire, M.T., Seagle, H. M. and Hill, J.O. (1998) Psychological symptoms in individuals successful at long-term maintenance of weight loss. *Health Psychology*, 17, 336-45.

Lacey, J., Coker, S, and Birtchnell, S. (1986) Bulimia: factors associated with its etiology and maintenance, *International Journal of Eating Disorders*, 5, 475-487.

Laessle, R., Platte, P., Schweiger, U., & Pirke, K.M. (1996) Biological and psychological correlates of intermittent dieting behaviour in young women: A model for bulimia nervosa. *Physiology and Behaviour*, 60, 1-5.

Langlie, J. (1977) Social events health beliefs, and preventive health behaviours, *J Health Soc Behav*, 18, 244-60.

Lattimore, P. (2001) Stress –induced eating: an alternative method for inducing ego-threatening stress. *Appetite*. 36, 187-188.

Lattimore. P. and Caswell, N. (2004) Differential effects of active and passive stress on food intake in restrained and unrestrained eaters. *Appetite*. 42. 167-173.

Laudat, M., Cerdas, S., Fournier, C. (1988) Salivary cortisol measurement: a practical approach to assess pituitary adrenal function. *Journal of Clinical Endocrinol Metab.* 66. 343-348.

Lazarus, R., Folkman, S. (1984) *Stress, Appraisal, and coping*. New York: Springer.

Lazarus, R. (1991) *Emotion and Adaptation*. Oxford: Oxford University Press.

Leibel, R. L., Chung, W. K. and Chua, S. C. Jr (1997) The molecular genetics of rodent single gene obesities. *Journal of Biological Chemistry*, 272, 31937-31940.

Leibowitz, S.F. (1978). Paraventricular Nucleus: A primary site mediating adrenergic stimulation of feeding and drinking. *Pharmacology Biochemistry & Behaviour*: 8: 163-175.

Leibowitz, S.F. (1980). Administration of 8-OH-DPAT into the midbrain raphe nuclei: effects on medial hypothalamic NE-induced feeding. *American Journal of Physiology*: 266:R1645-51.

(Leibowitz, S.F. 1988). Hypothalamic paraventricular nucleus: interaction between alpha 2-noradrenergic system and circulating hormones and nutrients in relation to energy balance. *Neuroscience and Biobehavioral Reviews*: 12:101-9.

Leibowitz, S.F. (1992) Neurochemical-neuroendocrine systems in the brain controlling macronutrient intake and metabolism. *Trends in Neurosciences.* 15:491-7.

Leibowitz, S.F., Sladek, C. Spencer, L. and Tempel, D. (1988). Neuropeptide Y epinephrine and norepinephrine in the paraventricular nucleus: stimulation of feeding and release of corticosterone, vasopressin and glucose. *Brain Research Bulletin*: 21:905-912.

Leibowitz, S.F. and Alexander, J.T. (1991). Analysis of neuropeptide Y induced feeding: dissociation of Y1 and Y2 receptor effects on natural meals patterns. *Peptides*:12.1251-1260.

Levine, K. and Ursin, H. (1991) What is stress? In: Brown. M. R., Rivier. C. and Koob. G. (Ed) *Stress, Neurobiology and Neuroendocrinology.* Marcel Decker. New York. 3-21.

Lewis, J. and Elder, P. (1985) An enzyme-linked immunosorbent assay (ELISA). *Journal of Steroid Biochemistry*. (22). 673-676.

Lingswiler, V., Crowther, J., Stephens, M. (1989) Emotional and somatic consequences of binge episodes. *Addictive Behaviour*. 14. (5). 503-11.

Lowe, M. R., & Fisher, E. B. (1983) Emotional Reactivity, emotional eating, and obesity: A naturalistic review. *Journal of Behavioural Medicine*, 6, 135-149.

Lowe, M.R. (1993) The effects of dieting on eating behaviour. A three factor model. *Psychological Bulletin*, 114, 100-121.

Luce, K., and Crowther, J. (1999) The reliability of the eating disorder examination—self-report questionnaire version (EDE-Q). *International Journal of Eating Disorders*, 25, 3, 349-351

McCann, B, S., Warnick, G, R., Knopp, R, H. (1990) Changes in plasma lipids and dietary intake accompanying shifts in perceived workload and stress. *Psychosomatic Medicine*. 52, 97-108.

McCartan BE, Lamey PJ, Wallace AM. (1996) Salivary cortisol and anxiety in recurrent aphthous stomatitis. *J Oral Pathol Med*, 25(7):357-359.

McConway MG, Chapman RS. (1986) Development and evaluation of a simple, direct, solid-phase radioimmunoassay of serum cortisol from readily available reagents. *Clin Chim Acta*, 158(1):59-70.

McEwen, B., Sakai, R., Spencer, R. (1993) Adrenal steroid effects on the brain: versatile hormones with good and bad effects. *Hormonally induced changes in mind and Brain*. 157-189.

Skaff, M., M. Pearlin, L. I., and Mullan, J. T. (1996) Transitions in the care giving career: Effects on sense of mastery. *Physiology and Ageing*. 11. 247-257.

McKenna RJ. (1972) Some effects of anxiety level and food cues on the eating behavior of obese and normal subjects: a comparison of the Schachterian and psychosomatic conceptions. *J Pers Soc Psychol*. Jun;22(3):311-9.

McLean, J.A., Barr, S.I. and Prior, J. C. (2001) Cognitive dietary restraint is associated with higher urinary cortisol excretion in healthy premenopausal women. *American Journal of Clinical Nutrition*. 73, 7-12.

McManus, F. and Waller, G. (1995) A functional analysis of binge eating. *Clinical Psychology Review*. 15, 845-863.

Macht, M. (1996). Effects of High and low energy meals on hunger, physiological processes and reactions to emotional stress. *Appetite*. 26(1), 71-88

Macht, M., Haupt, C., and Ellgring, H. (2005) The perceived function of eating is changed during examination stress: a field study, *Eating Behaviours*, 6, 109-112.

Macht, M. and Simons, G. (2000) Emotions and eating in everyday life, *Appetite*. 35, 65-71.

Maes, H. H., Neale, M. C., and Eaves, L.J. (1997) Genetic and environmental factors in relative body weight and human adiposity. *Behaviour Genetics*, 27, 325-351.

Maffei, M., Halaas, J., Ravussin, E., Pratley, R. E., Lee, G. H., Zhang, Y. Fei, H. Kim, S. Lallone, R. Ranganathan, S. et al. (1995) Leptin levels in human and rodent: measurement of plasma leptin and ob RNA in obese and weight-reduced subjects. *Nature Medicine,* 1,1155-61.

Margetts, B.M., Cade, J.E. and Osmond, C. (1989) Comparison of a food frequency questionnaire with a diet record. *International Journal of Epidemiology*, 18, 868-873.

Michalak, E.E., Tam, E.M.,Manjunath, C.V., Yatham, L.N., Levitt, A.J., Levitan, R.D. and Lam, R.W. (2004) Hard times and good friends: negative life events and social support in patients with seasonal and nonseasonal depression. *Canadian Journal of Psychiatry*, 49, (6), 408-411

Michaud, C, L., Kahn, J, P., Musse, N., Burlet, C., Nicolas, J, P., & Mejean, J. (1990). Relationships between a critical life event and eating behaviour in high school students. *Stress Medicine*. 6, 57-64.

Mond, J., Hay. P. J., Rodgers. B., Owen. C. and Beumont, P. (2004) Validity of the eating Disorder Examination Questionnare (EDE-Q) in screening for eating disorders in community samples. *Behaviour Research and Therapy*, (42), 551-567.

Morley J.E. and Blundell, J.E. (1988). The neurobiological basis of eating disorders:Some formulations. Biological Psychiatry

Morley, J.D., & Levine, A.S. (1980). Stress-induced eating is mediated through endogenous opiates. *Science*, 209, 1259-1261.

Morley JE, Levine AS, Rowland NE.(1983) Minireview. Stress induced eating. *Life Sci*. May 9;32(19):2169-82.

Morley, J.E., Levine, A.S. and Willenbring, M.L. (1986). Stress-induced feeding disorders. In: Carruba, M.O. and Blundell, J.E. (eds.): Pharmacology of Eating Disorders: Theoretical and Clinical Development. Raven Press, New York.

Motz, J.H. and Macdonald, J. (1985). Neuropeptide Y: Direct and indirect action on insulin secretion in the rat. *Peptides*: 6,1155-1159.

Mueller, W. M., Gregoire, F. M., Stanhope, K. L., Mobbs, C, V., Mizuno, T. M., Warden, C. H., Stern, J. S. and Havel, P. J. (1998) Evidence that glucose metabolism regulates leptin secretion from cultured rat adipocytes. *Endocrinology*, 139, 551-558.

National Heart, Lung, and Blood Institute (1998) Clinical Guidelines on the identification, evaluation and treatment of overweight and obesity in adults: the evidence report. *Obesity Research*, 6 (suppl). 2), 51S-210S.

Neel, J. (1982) The thrifty genotype revisited. In *The Genetics of Diabetes Mellitus,* eds. J. Köbberling and R. Tattersall, pp. 137-47. New York: Academic Press.

Neel, J. (1962) Diabetes mellitus: A "thrifty" genotype rendered detrimental by "progress"? *Am. J. Hum. Genet.* 14:353-62.

Nemeroff, C. B., Osbar, A.J., III, Bissette, G,. Jahnke, G,. Lipton, A. & Prange, A., Jr. (1978). Cholecystokinin inhibits tail pinch-induced eating in rats, *Science*, 200, 793-794.

Nielsen, S. J., Siega-Riz, A. M., and Popkin, B. M. (2002) Trends in energy intake in U. S. Between 1977 and 1996: similar shifts seen across age groups. *Obesity Research,* 10, 370-8.

Nobrega, J. N., Dixon, L. M., Troncone, L. R. P. & Barros, H. T. (1989). Effects of chronic haloperidol on stress-induced oral behaviour in rats. *Psychopharmacology*, 98, 476-482.

Ogden, J. (1993) The measurement of restraint: Confounding success and failure? *International Journal of Eating Disorders*. 13, 69-76.

Ogden, J. (2003). *The psychology of eating: from healthy to disordered behaviour.* Oxford: Blackwell Publishing

Okada, S., York, D., Bray, G., (1992) Mifiprestone (RU 486) a blocker of type II glucocorticoid and progestin receptors, reverses a dietary form of obesity. *American Journal of Obesity* 262, R1106-R1110.

Oliver, G., and Wardle, J. (1999). Perceived effects of stress on food choice. *Physiol. & Behav.* 66(3), 511-515.

Oliver, G., Wardle, J., and Gibson, E, L. (2000). Stress and food choice: A laboratory study. *Psychosomatic Medicine*, 62, 853-65.

Pajak, S., Mears, L., Kendall, T., Katona. C. and Medina, J. (2003) Workload and workload patterns in consultant psychiatrists. Workforce planning team, The Royal College of Psychiatrists. London.

Pearlin, L. and Schooler, C. (1978) The structure of coping. *Journal of Health and Social Behaviour*, 19, 2-21.

Pearlin, L., Menaghan, E., Lieberman, M. and Mullan, J. (1981) The Stress Process. *Journal of Health and Social Behaviour*. 22. Dec. 337-356.

Pernow, J. Lundberg,J.M., Kauser, I. et al (1986). Plasma neuropeptide Y like immunoreactivity and catecholamines during various degrees of sympathetic activation in man. *Clinical Physiology*., 6:561-578.

Peruzzo, B., Pastor, F.E., Blazquez, J.L., Schobitz, K., Paleaz, B., Amat, P. et al (2000). A second look at the barriers of the medial basal hypothalamus. *Experimental Brain Research*. 132,10-26.

Philip, W. and James, T. (2002) A world view of the obesity problem. In: Fairburn C. G., and Brownell, K. D. (Eds), London. *Eating Disorders and Obesity: a comprehensive handbook*. The Guilford Press.

Plotsky, P.M., Cunningham, Jr, E.T. and Widmaier, E.P. (1989) Catecholaminergic modulation of corticotrophin-releasing factor and adrenocorticotropin secretion. *Endocr Rev*. 10:437-458.

Plotsky, P.M. and Meaney, M.J. (1993) Early postnatal experience alters hypothalamic corticotropin-releasing factor (CRF) mRNA, median eminence CRF content and stress induced release in adult rats. *Brain Research and Molecular Brain Research*, 18, 195-200.

Pollard, T, M., Steptoe, A., Canaan, L., Davies, G, J., Wardle , J. (1995). Effects of academic examination stress on eating behavior and blood lipid levels. *International Journal of Behavioral Medicine*, 2, 299-320.

Polivy, J. (1996). Psychological consequences of food restriction. *Journal of the American Dietetic Association*, 96, 589-594.

Polivy, J. and Herman, C. (1985) Dieting and bingeing: A causal analysis. *American Psychologist*. 40. 193-201.

Polivy, J. and Herman, C. (1999) Distress and eating: Why do dieters overeat? *International Journal of Eating Disorders*. 26. 153-164.

Polivy, J., & Herman, C.P. (1999). The effects of resolving diet on restrained and unrestrained eaters: "The false hope syndrome." *International Journal of Eating Disorders*, 26, 434-447.

Polivy, J., & Herman, C.P., & McFarlane, T. (1994). Effects of anxiety on eating: does palatability moderate distress induced overeating in dieters. *J Abnorm Psychol*. 103, 505-10.

Polonsky, K. S., Given, B. D. and VanCauter, E. (1988) Twenty-four hour profiles and pulsatile patterns of insulin secretion in normal and obese subjects. *J. Clin. Invest*, 81, 442-448.

Prentice, A. M. (2001) Overeating: The health risks. *Obesity Research*, 9, 234S-238S.

Pyle, R., Mitchell, J., Eckert, E. (1981) Bulimia: A report of 34 cases. *Journal of Clinical Psychiatry*.42. 60-64.

Rand, C., Stunkard, A. (1978) Obesity and psychoanalysis. *American Journal of Psychiatry*. 135 (5) 547-51.

Renie, K. L., and Jebb, S. A. (2005) National Prevalence of Obesity: Prevalence of obesity in Great Britain, *Obesity Reviews*. 6, 11-12.

Rijsdik, F.V., Sham, P.C., Sterne, A. Purcell, S. McGuffin, P. Farmer, A. Goldberg, D. Mann, A. Cherny, S.S., Webster, M. Ball, D. Eley, T.C. and Plomin, R. (2001) Life events and depression in a community sample of siblings. *Psychological Medicine*. 31, 401-410.

Ritter, S., Watts, A.G., Dinh, Thu.T., Sanchez-Watts, G. and Pedrow, C. (2003) Immunotoxin lesion of hypothalamically projecting norepinephrine and epinephrine neurons differentially affects circadian and stressor stimulated corticosterone secretion. *Endocrinology*. 144: 13357-1367.

Robbins, T. W., & Fray, P. J. (1980). Stress induced eating: fact, fiction or misunderstanding? *Appetite*, 1, 103-33.

Robertson, W.R., Lambert, A and Loveridge, N. (1987). The role of modern bioassays in clinical endocrinology. *Clin. Endocrinol.*, 27: 259-278.

Roland, C.R., Bhakthavatsalam, P. and Leibowitz, S.F. (1986) Interaction between corticosterone and alpha-2-noradrenergic system of the paraventricular nucleus in relation to feeding behaviour. *Neuroendocrinol.,*42: 296-305.

Ruderman, A. (1983) The restraint Scale: A psychometric investigation. *Behaviour Research and Therapy,* 21. 253-258.

Ruggiero, D.A., Ross, C.A., Anwar, M, Park, D.H., Joh, T.H. and Reis, D.J. (1985) Distribution of neurons containing phenylethanolamine N-methyltransferase in medulla and hypothalamus of rat. *Journal of Comparative Neurology*., 239: 127-154.

Rutledge, T., & Linden, W. (1998). To eat or not to eat: affective and physiological mechanisms in the stress eating relationship. *J. of Behav. Med.* 21(2), 221-240.

Sapolsky, R. M., Romero, L. M. and Munck, A. U. (2000) How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory and preparative action. *Endocrine Reviews*. 21(1): 55-89.

Schachter, S., Goldman, R., & Gordon, A., (1968). Effects of fear, food deprivation and obesity on eating. *Journal of Personality and Social Psychology*, 10, 91-97.

Schachter, S. (1971). Some extraordinary facts about obese humans and rats. *American Psychologist*, 26, 129-144

Schachter, S., Goldman, R., & Gordon, A. (1968). Effects of fear, food deprivation and obesity on eating. *Journal of Personality and Social Psychology*, 10, 91-97.

Schlundt, D. G., Taylor, D., Hill, J. O., Sbrocco, T., Pope-Cordle, J., Kasser, T., & Arnold, D. (1991). A behavioural taxonomy of obese female participants in a weight-loss program. *American Journal of Clinical Nutrition*, 53, 1151-1158.

Schmidt, U., Tiller, J., Andrews, B., Blanchard, M., and Treasure, J. (1997) Is there a specific trauma precipitating the onset of anorexia nervosa? *Psychological Medicine,* 27, 523-530.

Schwartz, M. W., Woods, S. C., Porte, D. Seeley, R. J. and Baskin, D. G. (2000) Central nervous system control of food intake. *Nature*, 404. 661-671.

Seyle , H. (1951) *The physiology and pathology of exposure to stress*. Montreal. Acta.

Shapiro, J. R. and Anderson, D.A. (2005) Counterregulatory eating behaviour in multiple item test meals. *Eating Behaviours*, 6, 169-178.

Siegal, P. S. & Brantley, J. J. (1951). The relationship of emotionality to the consummatory response of eating. *Journal of Experimental Psychology*, 42, 304-306.

Sindelar, D. K., Havel, P. J., Seeley, R. J., Wilkinson, C. W., Woods, S. C. and Schwartz, M. W. (1999) Low plasma leptin levels contribute to diabetic hyperphagia in rats. *Diabetes*, 48. 1275-1280.

Slochower, J. (1976). Emotional labelling and overeating in obese and normal weight individuals. *Psychsomatic Medicine*, 38, 131-139.

Slochower, J. (1983). *Excessive eating: the role of emotions and environment*. New York: Human Sciences Library.

Slochower, J., & Kaplan, S. P. (1980). Anxiety, perceived control, and eating in obese and normal weight persons. *Appetite*, 1, 75-83.

Slof, R. Mazzeo, S. Bulik, C. (2003) Characteristics of women with persistent thinness. *Obesity Research*, 11, 971-977.

Snaith R. P. and Zigmond A. S. (1994) The Hospital Anxiety and Depression Scale Manual. NFER. Nelson, Windsor.

Spitzer, L., and Rodin, J. (1983) Arousal-induced eating; Conventional wisdom or empirical finding? In J, Cappioco, and R, Pette. (eds), *Social Psychophysiology* New York Guilford Press. 565-591.

Stanley, B.G. (1993). Neuropeptide Y in multiple hypothalamic sites controls eating behaviour, endocrine and autonomic systems for body energy balance. In W.F.

Colmers, & C. Wahlestedt (Eds), *The biology of neuropeptide Y and related peptides*, pp. 457-509. Totowa, N.J.: Humana Press

Stanley, B.G. Anderson, K.C. Grayson, M.H. and Leibowitz, S.F. (1989). Repeated hypothalamic stimulation with neuropeptide Y increases daily carbohydrate and fat intake and body weight gain in female rats. *Physiology of behaviour*:46.173-177.

Stanley, B.G., Daniel, D.R., Chin, A.S. and Leibowitz, S.F. (1985). Paraventricular injections of peptide YY and neuropeptide Y preferentially enhance carbohydrate ingestion. *Peptides*:6:1205-1211.

Stanley, B.G., Kyrkouli, S.E., Lampert, S. and Leibowitz, S.F. (1986). Neuropeptide Y chronically injected into the hypothalamus: a powerful neurochemical inducer of hypophagia and obesity. *Peptides*., 7:1189-92.

Steckler, T., Holsboer, F.(1999) Corticotropin-releasing hormone receptor subtypes and emotion. *Biol Psychiatry*. 1;46 (11):1480-508.

Stice, E. (2000) *Sociocultural influences on body image and eating disturbance*, In, *Eating disorders and obesity: a comprehensive handbook*, (2nd ed), Eds Fairburn, C.G. and Brownell, K,D. (2000), The Guilford Press, New York, 103-107.

Striegel-Moore, R.; Silberstein, L., Frensch, P. (1989) A prospective study of disordered eating among college students *International Journal of Eating Disorders,* 8(5), Sep. pp. 499-509.

Striegel_Moore, R. H., Wilson, G. T., Wilfley, D.E., Elder, K.A., and Brownell, K.D. (1998) Binge eating in an obese community sample. *International Journal of Eating Disorders*, 23, 27-37.

Stone , A, A., & Brownell, K, D. (1994). The stress-eating paradox: Multiple daily measurements in adult males and females. *Psychology and Health*, 9, 425-36.

Stunkard, A.J. and Messick, S. (1985) The three factor eating questionnaire to measure dietary restraint, disinhibition, and hunger. *Journal of Psychosomatic Research*. 29, 71-81.

Stunkard, A. J. and Sobal, J. (1995) Psychosocial consequences of obesity. In: Brownell, K. D., Fairburn, C. D. eds. *Eating Disorders and Obesity: A Comprehensive Handbook.* New York: Guilford Press, 417-21.

Takahashi, N. Patel, H, R., Qi, Y., Dushay, J., Ahima, R, S. (2002) Divergent effects of leptin in mice susceptible or resistant to obesity. *Hormone & Metabolic Research.* 34, 691-697.

Tanofsky-Kraff, M., Wilfley, D.E. and Spurrell, E. (2000) Impact of interpersonal and ego-related stress on restrained eaters. *International Journal of Eating Disorders*, 27, 411-418.

Tataranni, P., Larson, D., Snitker, S., Young, J. et al (1996)Effects of glucocorticoid on energy metabolism and food intake in humans. *American Journal of Physiology. 271, E317-E325.*

Telch, C.F. and Agras, W.S. (1994) Obesity, binge eating and psychopathology: are they related? *International Journal Eating Disorders.*15, 53-61.

Telch, C.F. and Stice. E. (1998) Psychiatric comorbidity in women with binge eating disorder: prevalence rates from a non-treatment-seeking sample. *Journal of Consulting Clinical Psychology*, 66, 768-76.

Tempel, D.L. and Leibowitz, S.F. (1993) Glucocorticoid receptors in the PVN: interactions with NE, NPY and Gal in relation to feeding. *American J Physiol.*, 265:E794-E800.

Tempel, D., Leibowitz, S. (1994) Adrenal Steroid receptors: interactions with brain neuropeptide systems in relation to nutrient intake and metabolism. *Journal of Neuroendocrinology.*6 479-501.

Thompson, J.K., Heinberg, L.J., Altabe, M., and Tantleff-Dunn, S. (1999) *Exacting beauty: theory, assessment, and treatment of body image disturbance*. Washington, DC: American Psychological Association.

Treasure, J.L. and Holland, A.J. (1995). Genetic factors in eating disorders. In: Szmukler, G., Dare, C. and Treasure, J. (Eds.). Handbook of Eating Disorders. Theory, Treatment and Research. John Wiley and Sons, Chichester.

Treasure, J. and Owen, J. (1997) Intriguing links between animal behavior and anorexia nervosa. *Int J Eat Disord*. 21(4):307-11.

Troop, N., and Treasure, J. (1997) Psychosocial factors in the onset of eating disorders: Responses to life events and difficulties. *British Journal of Medical Psychology*, 70, 373-385.

Tucker, D.C., Saper, C.B., Ruggiero, D.A. and Reis, D.J. (1987) Organisation of central adrenergic pathways. I. Relationships of ventrolateral medullary projections to the hypothalamus and spinal cord. *Journal of Comparative Neurology*., 259: 591-603.

Ursin, H. (1980) *Coping and Health*. Plenum Press. 259-279.

Ursin, H. (1998) The psychology in psychoneuroendocrinology. *Psychoneuroendocrinology*. 23.6. 555-570

Van Strein, T. (1999). Success and failure in the measurement of restraint: Notes and data. *Int. J. Eat. Dis*. 25, 441-9.

Valdez, R. and Williamson, D. F. (2002) Prevalence and demographics of obesity. In: Fairburn C.G, Brownell, K.D. Eds, *Eating Disorders and Obesity: A comprehensive Handbook.* New York: Guildford; 2002, pp 417-21.

de Waal, F.B.M. (2002). Evolutionary psychology: The wheat and the chaff. *Psychological Science*, 11, 187-191.

Wadden, T. A., and Stunkard, A. J. (2002) Handbook of obesity treatment. (Eds), The Guilford Press. New York.

Wallis, D. J. and Hetherington, M.M. (2004) Stress and eating: the effects of ego threat and cognitive demand on food intake in restrained and emotional eaters. *Appetite*, 43, 39-46.

Walsh, B. T., Katz, J. L., Levin, J., *et al* (1978) Adrenal activity in anorexia nervosa. Psychosom.Med., 40, 499-506.

Wang, J. Liu, R. Hawkins, M. Barzilai, N. And Rossetti, L. A. (1998) A nutrient-sensing pathway regulates leptin gene expression in muscle and fat. *Nature,* 393, 684-688.

Ward, A., Brown, N., Lightman, S., *et al* (1998) Neuroendocrine, appetitive and behavioural responses to d-fenfluramine in women recovered from anorexia nervosa. *Br.J.Psychiatry*, **172**, 351-358.

Wardle, J., Steptoe, A., Oliver, G., & Lipsey, Z. (2000). Stress dietary restraint and food intake. *J. of Psych. Res*. 48(2), 195-202.

Wardle, J. and Gibson, E. L. (2001) *Impact of stress on diet: processes and implications*. In: Standfield, S. Marmot, M. G. eds. *Stress and the heart*, UK: BMJ Books.

Wardle, J., Waller, J, & Rapoport, L. (2001) Body dissatisfaction and binge eating in obese women: the role of Restraint and depression. *Obesity Research*, 9, 778-787.

Weidner, G., Kohlmann, C, W., Dotzauer, E., Burns, L, R. (1996). The effects of academic stress on health behaviors in young adults. *Anxiety Stress Coping*, 9, 123-33.

Wellberg, L.A.M. and Seckl, J.R. (2001) Prenatal stress, glucocorticoids and the programming of the brain. *Journal of Neuroendocrinology*, 13, 113-128.

Westenhoefer, J. (1991) Dietary restraint and disinhibition: Is restraint a homogenous construct? *Appetite*. 16. 45-55.

Westenhoefer, J., Stunkard, A., & Pudel. (1999). Validation of the flexible and rigid control dimensions of dietary restraint. *International Journal of Eating Disorders*, 26, 53-64.

Whitnall, M,H. (1993) Regulation of the hypothalamic corticotrophin-releasing hormone neurosecretory system. *Progress Neurobiol.* 40: 573-629.

Wilchek, M. and Bayer, E.(1990) *Methods in molecular biology, Vol1: Proteins.* Humana Press, Clifton New Jersey.

Willenbring, M.L., Levine, A.S. and Morley, J.E. (1986). Stress-induced eating: a pilot study. *International Journal of Eating Disorders* 5, 855 - 864.

Willox, J., Corr, J., Shaw, J., Richardson, M., Calman, K. (1984) Prednisolone as an appetite stimulant in patients with cancer. *British Medical Journal*. 288. 27

Woods, S. Lotter, E. McKay, L. and Porte, D. J. (1979) Chronic intracerebroventricular infusion of insulin reduces food intake and body weight of baboons. *Nature*, 282, 503-505.

World Health Organization (1998) *Obesity, preventing and managing the global epidemic*, Geneva.

Yacono-Freeman, L. M. and Gil. K. M. (2004) Daily stress, coping, and dietary restraint in binge eating. *International Journal of Eating Disorders*. 36, 204-212.

York, D, A. (1992). Central regulation of appetite and autonomic activity by CRH, glucocorticoids and stress. *Progress Neuroendocrinimmunol*, 5, 153-65.

Zarjevski, N. Cusin, I. Vettor, R. Rohner-Jeanrenaud, F. and Jeanrenaud, B. (1993). Chronic intracerebroventricular neuropeptide Y administration to normal rats mimics hormonal and metabolic changes of obesity. *Endocrinology*:133:1753-178.

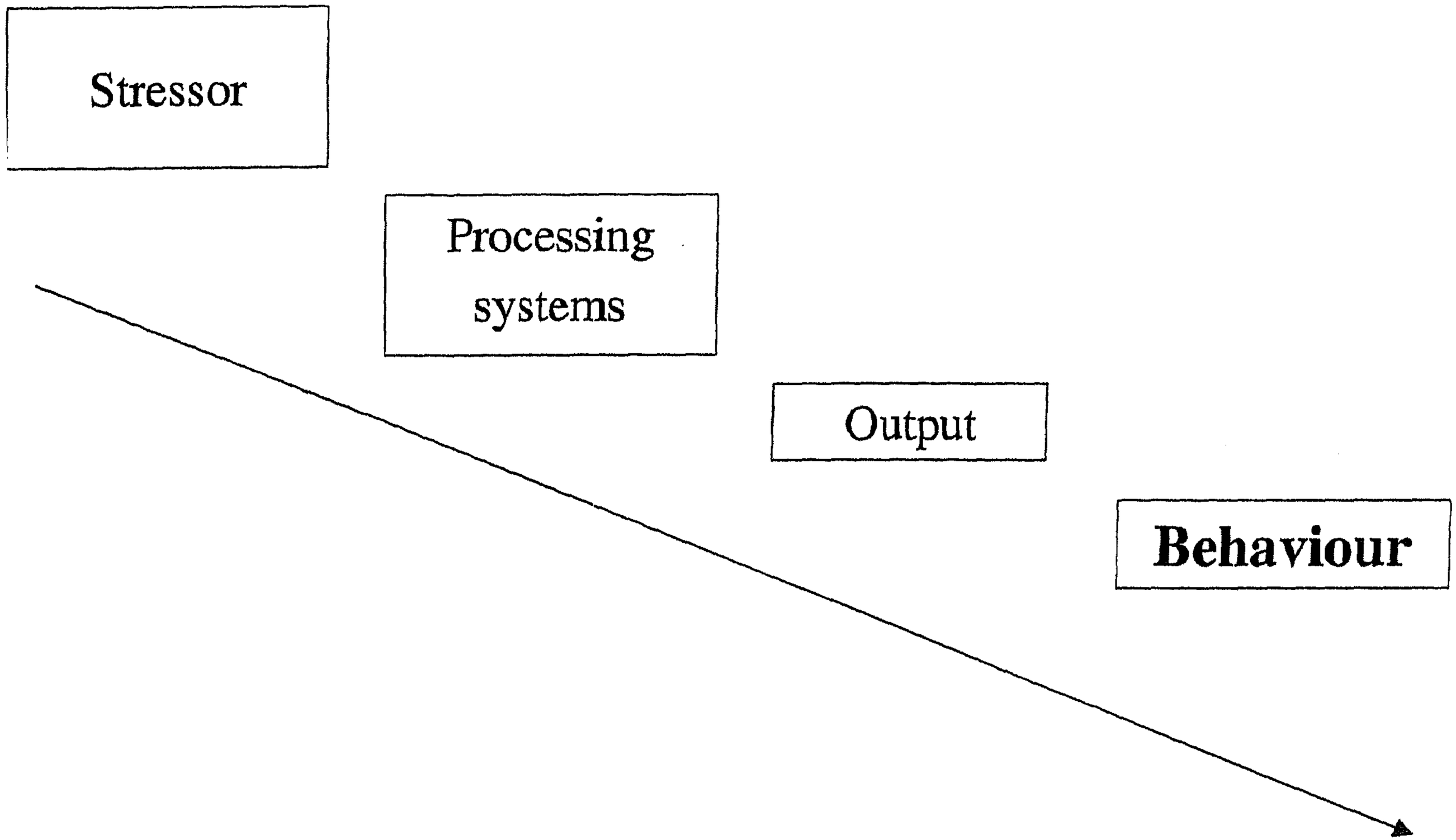
Zigmond, A. and Snaith, R. (1983) The hospital anxiety and depression scale. *Acta Psychiatrica Scandanavica*, 67. 361-370.

Zumoff, B., Walsh, B. T., Katz, J. L., *et al* (1983) Subnormal plasma dehydroisoandrosterone to cortisol ratio in anorexia nervosa: a second hormonal parameter of ontogenic regression. *J.Clin.Endocrinol.Metab*, 56, 668-672.

List of Appendices.

A transactional theory of behaviour

Lazarus & Folkman (1984)



Appendix 2

Activation Theory Levine and Ursin (1991).

Activation Theory

Input

Stress :Stimulus

e.g Psychosocial

Environmental

Physical

Processing Systems+Subjective Experience

Psychological Filters: Mastery:Coping

Output

ANS: effecting CVS, Respiratory system etc.

HPA Axis - Cortisol: Adrenaline

Appendix 3

Mastery Scale	Pearlin (1981).

Mastery Scale

How strongly do you agree or disagree with these statements about yourself?

Please circle one response

1. There is really no way I can solve some of the problems I have.
   Strongly Disagree/ Disagree/ Agree/ Strongly Agree

2. Sometimes I feel that I'm being pushed around in life.
   Strongly Agree/ Agree/ Disagree/ Strongly Disagree

3. I have little control over the things that happen to me.
   Strongly Disagree/ Disagree/ Agree/ Strongly Agree

4. I can do just about anything I really set my mind to.
   Strongly Agree/ Agree/ Disagree/ Strongly Disagree

5. I often feel helpless in dealing with the problems of life.
   Strongly Disagree/ Disagree/ Agree/ Strongly Agree

6. What happens to me in the future mostly depends on me.
   Strongly Agree/ Agree/ Disagree/ Strongly Disagree

7. There is little I can do to change many of the important things in my life.
   Strongly Disagree/ Disagree/ Agree/ Strongly Agree

Pearlin, L. Menaghan, E. Lieberman, M. and Mullan, J. (1981).

Appendix 4

List of Threateneing Experiences (Brugha et al 1985).

The List of Threatening Experiences Questionnaire (LTEQ)

Over the past 6 months

Life events category	Yes	No
You yourself suffered a serious illness, injury or an assault.	☐	☐
A serious illness, injury or assault happened to a close relative	☐	☐
Your parent child or spouse died.	☐	☐
A close family friend or another relative (aunt cousin, grandparent) died.	☐	☐
You had a separation due to marital difficulties.	☐	☐
You broke off a steady relationship.	☐	☐
You had a serious problem with a close friend, neighbour or relative.	☐	☐
You became unemployed or you were seeking work unsuccessfully.	☐	☐
You were sacked from your job.	☐	☐
You had a major financial crisis.	☐	☐
You had problems with the police and a court appearance.	☐	☐
Something you valued was lost or stolen.	☐	☐
At least one such event in the past six months.	☐	☐
At least one such event in the past three months.	☐	☐

Brugha, T. Bebbington, P, Tennant, C. and Hurry, J. (1985).

Appendix 5

General Health Questionnaire – 12 (Goldberg 1972)

GHQ-12

We would like to know if you have had any medical complaints and how your health has been in general over the past week.
Please answer ALL the questions on the following page by circling the answer you think most closely applies to you.
Remember that we want to know about present and recent complaints, not those you had in the past.
It is important that you answer ALL the questions

HAVE YOU RECENTLY:

1. ...been able to concentrate on whatever you're doing?	Better than usual	Same as usual	Less than usual	Much less than usual
2. ...lost much sleep over worry?	Not at all	No more than usual	Rather more than usual	Much more than usual
3. ...felt that you are playing a useful part in things?	More so than usual	Same as usual	Less useful than usual	Much less than usual
4. ...felt capable of making decisions about things?	More so than usual	Same as usual	Less so than usual	Much less capable
5. ...felt constantly under strain?	Not at all	No more than usual	Rather more than usual	Much more than usual
6. ...felt you couldn't overcome your difficulties?	Not at all	No more than usual	Rather more than usual	Much more than usual
7. ...been able to enjoy your normal day-to-day activities	More so than usual	Same as usual	Less so than usual	Much less than usual
8. ...been able to face up to your problems?	More so than usual	Same as usual	Less able than usual	Much less able
9. ...been feeling unhappy and depressed?	Not at all	No more than usual	Rather more than usual	Much more than usual
10. ...been losing confidence in yourself?	Not at all	No more than usual	Rather more than usual	Much more than usual
11. ...been thinking of yourself as a worthless person?	Not at all	No more than usual	Rather more than usual	Much more than usual
12. ...been feeling reasonably happy, all things considered?	More so than usual	Same as usual	Less so than usual	Much less than usual

Sex (please circle the correct response): **Male** **Age:** _____

Female

Appendix 6

Hospital Anxiety and Depression Scale (Zigmond and Snaith 1983).

HAD scale

1. I feel tense or "wound up":

Most of the time	A lot of the time	Occasionally	Not at all
☐	☐	☐	☐

2. I still enjoy the things I used to enjoy:

Definitely as much	Not quite so much	Only a little	Not at all
☐	☐	☐	☐

3. I get a sort of frightened feeling as if something awful is about to happen:

Very definitely and quite badly	Yes, but not too badly	A little, but it doesn't worry me	Not at all
☐	☐	☐	☐

4. I can laugh and see the funny side of things:

As much as I always could	Not quite so much now	Definitely not so much now	Not at all
☐	☐	☐	☐

5. Worrying thoughts go through my mind:

A great deal of the time	A lot of the time	From time to time	only occasionally
☐	☐	☐	☐

6. I feel cheerful:

Not at all	Not often	Sometimes	Most of the time
☐	☐	☐	☐

7. I can sit at ease and feel relaxed:

Definitely	Usually	Not often	Not at all
☐	☐	☐	☐

8. I feel as if I am slowed down:

Nearly all the time	Very often	Sometimes	Not at all
☐	☐	☐	☐

9. I get a sort of frightened feeling like "butterflies" in the stomach:

Not at all	Occasionally	Quite often	Very often
☐	☐	☐	☐

10. I have lost interest in my appearance:

Definitely	I don't take so much care as I should	I may not take quite as much care	I take just as much care as ever
☐	☐	☐	☐

11. I feel restless as if have to be on the move.

Very much indeed	Quite a lot	Not very much	Not at all
☐	☐	☐	☐

12. I look forward with enjoyment to things:

As much as I ever did	Rather less than I used to	Definitely less than I used to	Hardly at all
☐	☐	☐	☐

13. I get sudden feelings of panic:

Very often indeed	Quite often	Not very often	Not at all
☐	☐	☐	☐

14. I can enjoy a good book or ratio or TV programme:

Often	Sometimes	Not often	Very seldom
☐	☐	☐	☐

Appendix 7

Eating Disorder Examination-Questionnaire
(Fairburn and Beglin 1994).

EDE-Q4

Instructions.
The following questions are concerned with the PAST FOUR WEEKS ONLY (28 days). Please read each question carefully and circle the appropriate number on the right. Please answer all the questions.

ON HOW MANY DAYS OUT OF; THE PAST 28 DAYS	No days	1-5 days	6-12 days	13-15 days	16-22 days	23-27 days	Every day
1. Have you been deliberately trying to limit the amount of food you eat to influence your shape or weight?	0	1	2	3	4	5	6
2. Have you gone for long periods of time (8 hours or more) without eating anything in order to influence your shape or weight?	0	1	2	3	4	5	6
3. Have you tried to avoid eating any foods which you like in order to influence your shape or weight?	0	1	2	3	4	5	6
4. Have you tried to follow definite rules regarding your eating in order to influence your shape or weight; for example, a calorie limit, a set amount of food, or rules about what or when you should eat?	0	1	2	3	4	5	6
5. Have you wanted your stomach to be empty?	0	1	2	3	4	5	6
6. Has thinking about food or its calorie content made it much more difficult to concentrate on things you are interested in; for example, read, conversation?	0	1	2	3	4	5	6
7. Have you been afraid of losing control over eating?	0	1	2	3	4	5	6

ON HOW MANY DAYS OUT OF; THE PAST 28 DAYS	No days	1-5 days	6-12 days	13-15 days	16-22 days	23-27 days	Every day
8. Have you had episodes of binge eating?	0	1	2	3	4	5	6
9. Have you eaten in secret? (Do not count binges).	0	1	2	3	4	5	6
10. Have you definitely wanted your stomach to be flat?	0	1	2	3	4	5	6
11. Has thinking about shape or weight made it more difficult to concentrate on things you are interested in; for example read, watch TV or follow a conversation?	0	1	2	3	4	5	6
12. Have you had a definite fear that you might gain weight or become fat?	0	1	2	3	4	5	6
13. Have you felt fat?	0	1	2	3	4	5	6
14. Have you had a strong desire to loose weight?	0	1	2	3	4	5	6

OVER THE PAST FOUR WEEKS (28 DAYS)

15. On what proportion of times that you have eaten have you felt guilty because of the effect on your shape or weight? (do not count binges). (circle the number which applies).

0 - None of the times
1 - A few of the times
2 – Less than half the times
3 – Half the times
4 – More then half the times
5 – Most of the time
6 – Every time

16. Over the past four weeks (28 days), have there been any times when you have felt that you have eaten what other people would regard as an unusually large amount of food given the circumstances? (Please put appropriate number in the box).

0 – No

1 – Yes ☐

17. How many such episodes have you had over the past four weeks?

☐☐☐

18. During how many of these episodes of overeating did you have a sense of having lost control over your eating? ☐☐☐

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19. Have you had other episodes of eating in which you have had a sense of having lost control and eaten too much, but have <u>not</u> eaten an unusually large amount of food given the circumstances?

0 – No

1 – Yes ☐

20. How many such episodes have you had over the past four weeks?

☐☐☐

---

21. Over the past four weeks have you made yourself sick (vomit), as a means of controlling your shape or weight?

0 – No

1 – Yes ☐

22. How many times have you done this over the past four weeks?

☐☐☐

---

23. Have you taken laxatives as a means of controlling your shape or weight?

0 – No

1 – Yes ☐

24. How many times have you done this over the past four weeks?

☐☐☐

---

25. Have you taken diuretics (water tablets) as a means of controlling your shape or weight?

0 – No

1 – Yes ☐

26. How many times have you done this over the past four weeks?

☐☐☐

---

27. Have you exercised hard as a means of controlling your shape or weight?

0 – No

1 – Yes ☐

28. How many times have you done this over the past four weeks?

☐☐☐

OVER THE PAST FOUR WEEKS (28 DAYS) (Please circle the number which best describes your behaviour).	Not at all		Slightly		Moderately		Markedly
29. Has your weight influenced how you think about (judge) yourself as a person?	0	1	2	3	4	5	6
30. Has your shape influenced how you think about (judge) yourself as a person?	0	1	2	3	4	5	6
31. How much would it upset you if you had to weigh yourself once a week for the next four weeks?	0	1	2	3	4	5	6
32. How dissatisfied have you felt about your weight?	0	1	2	3	4	5	6
33. How dissatisfied have you felt about your shape?	0	1	2	3	4	5	6
34. How concerned have you been about other people seeing you eat?	0	1	2	3	4	5	6
35. How uncomfortable have you felt seeing your body; for example in the mirror, in shop window reflections, while undressing or taking a bath or shower?	0	1	2	3	4	5	6
36. How uncomfortable have you felt about others seeing your body; for example, in communal changing rooms, when swimming or wearing tight clothes?	0	1	2	3	4	5	6

Appendix 8

Food Frequency Questionnaire.
(Cade and Margetts; Margetts, Cade and Osmond, 1989).

Eating Questionnaire

CODE NUMBER: _____

Please read the following instructions carefully before completing the questionnaire

1. Please answer ALL the questions.

2. Select the answer that best applies to your situation and TICK the appropriate column.

3. Only tick ONE box on **each** line (i.e., one tick per food).

4. If you make a mistake, put a cross through the incorrect tick and then tick the correct answer.

For example:

How often do you eat the following foods?

		2 or more times a day	Every day	3-5 times a week	1-2 times a week	1-3 times a month	Rarely-never
Milk (including in tea or coffee)	Whole	–	–	–	–	–	–
	Semi-skimmed	–	–	–	–	–	–
	Skimmed	–	–	–	–	–	–

How often do you eat each of the following foods? (tick the appropriate box)
Please record what you normally eat and drink.

How often do you eat the following foods?

	2 or more times a day	Every day	3-5 times a week	1-2 times a week	1-3 times a month	Rarely -never
Milk (including in tea/coffee) :Whole	–	–	–	–	–	–
:Semi-skimmed	–	–	–	–	–	–
:Skimmed	–	–	–	–	–	–
Butter	–	–	–	–	–	–
Margarine (e.g., Stork, Clover)	–	–	–	–	–	–
Polyunsaturated margarine (e.g., Flora, sunflower)	–	–	–	–	–	–
Low fat spreads (e.g., Outline, Gold)	–	–	–	–	–	–
Ice cream	–	–	–	–	–	–
Yoghurt, Fromage Frais	–	–	–	–	–	–
Cheese (e.g., cheddar, cream cheese)	–	–	–	–	–	–
Low fat cheese (e.g., cottage, reduced fat cheese)	–	–	–	–	–	–
Eggs - fried	–	–	–	–	–	–
Eggs - not fried (boiled, poached, in baking)	–	–	–	–	–	–
Cheese and/or egg dishes	–	–	–	–	–	–

How often do you eat the following foods?

	2 or more times a day	Every day	3-5 times a week	1-2 times a week	1-3 times a month	Rarely -never
MEATS AND FISH						
Beef - roast/steak	–	–	–	–	–	–
Lamb - roast/chops	–	–	–	–	–	–
Pork - roast/chops	–	–	–	–	–	–
Chicken, turkey or other poultry	–	–	–	–	–	–
Bacon or gammon	–	–	–	–	–	–
Meat dishes (e.g., stew, curry, chilli)	–	–	–	–	–	–
Canned meats (e.g., corned beef, ham)	–	–	–	–	–	–
Meat pies, sausage rolls or pasties	–	–	–	–	–	–
Sausages or beef burgers	–	–	–	–	–	–
Liver, kidney, paté (other offal products)	–	–	–	–	–	–
Fish and seafood - not fried	–	–	–	–	–	–
- fried	–	–	–	–	–	–
- canned (e.g., tuna)	–	–	–	–	–	–
BREAD AND CEREALS						
White bread	–	–	–	–	–	–
Brown/granary bread	–	–	–	–	–	–
Wholemeal bread (including chapattis)	–	–	–	–	–	–
Sweet biscuits (plain and chocolate)	–	–	–	–	–	–
Crackers/crisp bread	–	–	–	–	–	–
Cakes/buns/pastries	–	–	–	–	–	–
Puddings (e.g., fruit pies, cheesecake)	–	–	–	–	–	–
Breakfast cereal:						
- High fibre (e.g., bran flakes, Weetabix)	–	–	–	–	–	–
- Ordinary (e.g., cornflakes, rice krispies)	–	–	–	–	–	–
- Muesli	–	–	–	–	–	–
Rice or pasta	–	–	–	–	–	–
FRUIT AND VEGETABLES						
Apples, pears	–	–	–	–	–	–
Oranges, grapefruit or other citrus fruit	–	–	–	–	–	–
Bananas	–	–	–	–	–	–
Green vegetables (e.g., cabbage, peas)	–	–	–	–	–	–
Carrots, tomatoes (fresh or canned)	–	–	–	–	–	–
Other vegetables (including salad vegetables)	–	–	–	–	–	–
Baked beans	–	–	–	–	–	–
Other beans/lentils (e.g., dahl)	–	–	–	–	–	–
Vegetable dishes (e.g., stew, curry - NO meat)	–	–	–	–	–	–

Potatoes - chips, roasted, fried	_	_	_	_	_	_
- not fried (boiled, baked)	_	_	_	_	_	_

How often do you eat the following foods?

	2 or more times a day	Every day	3-5 times a week	1-2 times a week	1-3 times a month	Rarely -never
BEVERAGES						
Beer or lager	_	_	_	_	_	_
Wine, sherry or spirits	_	_	_	_	_	_
Tea or coffee	_	_	_	_	_	_
Squash or fizzy drinks	_	_	_	_	_	_
Low calorie drinks	_	_	_	_	_	_
Pure fruit juices	_	_	_	_	_	_
MISCELLANEOUS						
Chocolate	_	_	_	_	_	_
Sweets	_	_	_	_	_	_
Sweet spreads (e.g., jam, marmalade)	_	_	_	_	_	_
Sugar (e.g., in tea/coffee, on cereal)	_	_	_	_	_	_
Crisps and savoury snacks	_	_	_	_	_	_
Nuts (including peanut butter)	_	_	_	_	_	_
Sauces and pickles (e.g., ketchup, Branston)	_	_	_	_	_	_
Salad oils, dressings, mayonnaise	_	_	_	_	_	_
_____	_	_	_	_	_	_
_____	_	_	_	_	_	_
_____	_	_	_	_	_	_

Are there any other foods that have not been recorded that you regularly eat?
Please write what they are and how often you eat them:

PLEASE COMPLETE THE FOLLOWING QUESTIONS ABOUT YOURSELF:

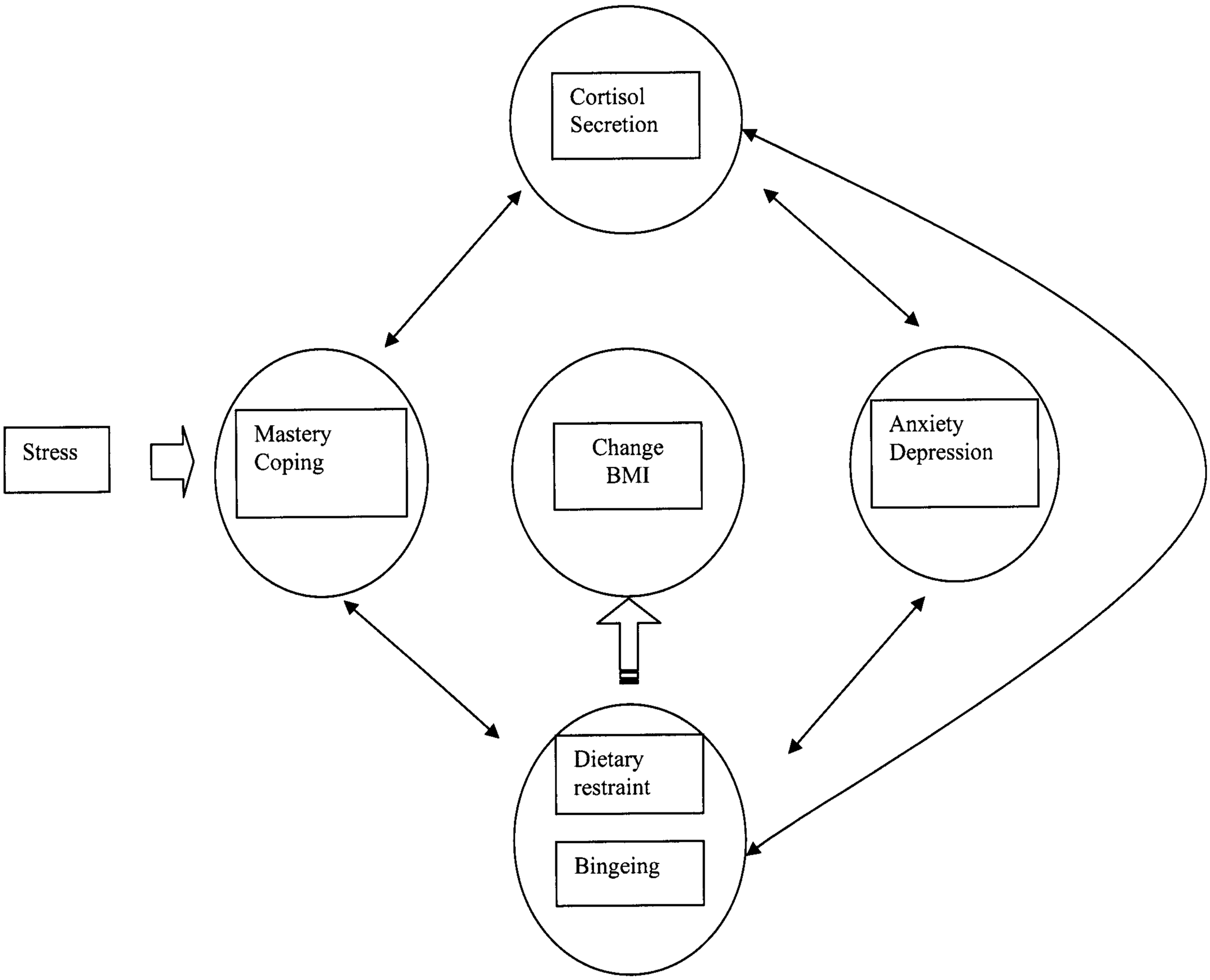
1. On the whole I would say that my feelings about eating a low fat diet are (circle one):

Generally favourable feelings	**Mixed feelings**	**Generally negative feelings**

Appendix 9

Spiral model showing association of variables in the person environment relationship and their involvement with change in body weight.

Appendix 9



Theoretical spiral association of variables in the person environment relationship and their involvement with change in body weight.